APPENDIX C

PREMEETING COMMENTS

CONTENTS

William Adams

I. Technical Issues Associated with a Water-column-Based Chronic Criterion

1. Besides selenite and selenate, which other forms of selenium in water are toxicologically important with respect to causing adverse effects on freshwater aquatic organisms under environmentally realistic conditions?

   The only form other than selenate and selenite in water (not sediment) that appears to be toxicologically important is selenocyanate. Little is known about the toxicity of this form, but it is found in petroleum and a limited number of mining discharges. Selenate and selenite are the predominant forms derived from mining, agricultural practices, fly ash, and natural shales. However, it is the conversion of these forms to organo-selenium compounds that are of most ecological relevance. Organo-forms are rarely found in water at detectable levels. However, it should be pointed out that the analytical methods for measuring these forms are not well developed.

2. Which form (or combination of forms) of selenium in water are most closely correlated with chronic effects on aquatic life in the field? (In other words, given current or emerging analytical techniques, which forms of selenium in water would you measure for correlating exposure with adverse effects in the field?) Note: Your response should include consideration of operationally defined measurements of selenium (e.g., dissolved and total recoverable selenium), in addition to individual selenium species.

   Given the state-of-the-art for measuring selenium in water and given that organo-forms of selenium are rarely found in the water column, the most relevant form of selenium to measure in water to correlate with adverse effects would be total recoverable selenium. This would include all forms of selenium in the water except a limited amount of non-bioavailable selenium that might be tied up in the crystalline structure of suspended solids. However, it should be pointed out that the body of literature is growing which shows that chronic effects in aquatic as well as terrestrial systems are primarily due to the conversion of inorganic forms to organic forms which are then taken up via the diet. This simply points to the fact that the diet is important and that chronic toxicity may not be principally due to waterborne selenium. Exceptions to this might be for primary producers.

3. A) In priority order, which water quality characteristics (e.g., pH, TOC, sulfate, interactions with other metals such as mercury) are most important in affecting

the chronic toxicity and bioaccumulation of selenium to freshwater aquatic life under environmentally realistic exposure conditions?

Overall, the eh (oxidative/reductive) state of an ecosystem is most important in determining the potential for chronic toxicity to occur because it significantly influences the formation of organo-forms of selenium. I do not believe we can prioritize these water quality characteristics due to insufficient information of their effects on expression of chronic toxicity. One could predict that, at the extremes, pH would be important due to speciation changes, but chronic data are not available to assess this. Sulfate appears unimportant in terms of the expression of chronic toxicity except potentially for primary producers.

B) Of these, which have been (or can be) quantitatively related to selenium chronic toxicity or bioaccumulation in aquatic organisms? How strong and robust are these relationships?

None, if one looks for a water quality characteristic which crosses all trophic levels. Sulfate would most likely correlate well with chronic toxicity of selenate to primary producers.

C) How certain are applications of toxicity relationships derived from acute toxicity and water quality characteristics to chronic toxicity situations in the field?

The applications of relationships derived from acute toxicity and water quality characteristics do not apply to chronic toxicity for most aquatic life (an exception to this might be the relationship between selenate and sulfate for algae). This is primarily due to the fact that acute toxicity is most often the result of water exposures whereas chronic effects are the result of selenium being incorporated into the diet where the predominant form of selenium is no longer an inorganic form.

II. Technical Issues Associated with a Tissue-Based Chronic Criterion

4. Which forms of selenium in tissues are toxicologically important with respect to causing adverse effects on freshwater aquatic organisms under environmentally realistic conditions and why?

Protein-bound (reduced forms) of selenium (i.e., most likely seleno-methionine or seleno-cysteine). Due to the lack of analytical methodologies to actually measure organo-forms of selenium little actual information on specific forms is available. However, there is a growing body of evidence that points to the fact that chronic toxicity is the result of the conversion of inorganic forms to organic forms either by plants or

animals in the water column or by microbes in the sediments under reducing conditions. These organo-selenium compounds enter the food chain and are transmitted to receptors where they mimic their sulfur analogs and interfere with normal metabolic processes. The conversion of inorganic forms to organic forms occurs through several rate limiting steps and at different rates under different environmental conditions. Understanding (and predicting) the factors controlling these rate limiting processes is the challenge of the next decade.

5. Which form (or combination of forms) of selenium in tissues are most closely correlated with chronic effects on aquatic life in the field? (In other words, given current or emerging analytical techniques, which forms of selenium in tissues would you measure for correlating exposure with adverse effects in the field?)

   At present, in spite of emerging techniques for organo-selenium forms, the best measure of selenium in tissues is total selenium. Measures of protein bound and organo-selenium would help advance the science.

6. Which tissues (and in which species of aquatic organisms) are best correlated with overall chronic toxicological effect thresholds for selenium?

   Measuring total selenium in fish ovaries (eggs) from gravid females (pre-spawning) appears to be a potentially useful monitoring tool. Certainty (uncertainty) in the threshold is unknown due to the lack of multiple data set. Values in the range of 8-12 ug/g have been proposed as a threshold.

7. How certain are we in relating water-column concentrations of selenium to tissue-residue concentrations in top trophic-level organisms such as fish? What are the primary sources of uncertainty in this extrapolation?

   Over the past few months my colleagues and I have endeavored to relate water-column concentrations of selenium to bird egg concentrations and tissue-residue concentrations in fish. For the water to bird egg analysis, we have developed a model based on a one step regression from mean water selenium to mean egg selenium concentration (Adams et al. 1997). This model represents the mean relationship over many sites, and the overall uncertainty and variability in applying a single model to different sites. The database we used was based on selenium egg and water concentrations from USFWS and USGS reports for 15 lentic sites in the western U.S. The sites covered a broad range of physical and ecological conditions. We believe this heterogeneity appropriately encompassed much or all of the uncertainty in applying such a model to any given site. Data for each site included different areas (e.g., Kesterson Reservoir within San Joaquin Valley) and different ponds (e.g., Pond #2 at

*Kesterson Reservoir) within an area. Our model allows us to quantify the uncertainty in relating water column selenium concentrations of mean egg residues. For purpose of example, we've run our model to predict the water concentration that would be protective of bird eggs with a selenium threshold of 8 – 20 mg/kg. Our analysis found that the 10th to 90th percentile range on the water concentration that would be protective of this endpoint, for **lentic** sites in the Western U.S., is 3 – 173 ug/L). Thus, the max:min uncertainty factor for the egg threshold is 2.5, whereas the 90:10 uncertainty factor for water is 58, or more than 20 times higher. This indicates that site variability is an important factor in extrapolating from water column selenium concentrations to tissue residue concentrations. Our model estimates that a water concentration of 5 ug/L is over-protective of 83% of sites of the sites examined (for the uncertain selenium threshold of 8 – 20 mg/kg). A water concentration of 2 ug/L would be over-protective of 94% f the sites for this uncertain egg threshold.*

Our efforts torelate water column and fish tissue selenium concentrations has met without much success, due to the absence of suitable data. The idea was to correlate the tissue concentrations (say fish eggs) to their diet and to the water column concentration of total recoverable selenium. This would be patterned after the approach used by Skorupa and Ohlendorf (1991) for birds. Our modeling efforts indicated a rather low degree of correlation due to the lack of good data sets where the appropriate information on dietary and selenium composition existed along with tissue and water selenium concentrations. This approach requires an extensive amount of data. For this approach to be useful it would have to be performed at several sites so the variability in the relationship could be assessed.

III. Technical Issues Associated with a Sediment-Based Chronic Criterion

8. Which forms of selenium in sediments are toxicologically important with respect to causing adverse effects on freshwater aquatic organisms under environmentally realistic conditions?

For the most part total selenium in bulk sediments has been used to relate sediment concentrations to possible effects. Recently, Van DerVeer and Canton (1997) demonstrated the importance of considering total organic carbon in sediments as a potential ligand for binding selenium, controlling its bioavailability or as a surrogate measure for the potential for inorganic forms to be converted to organic forms (actual mechanism was not explained in the publication). In general, it is thought that inorganic selenium is converted to organic forms in sediments by microorganisms under reducing conditions and that these organic forms are incorporated in the diet and are responsible for toxicity to higher trophic level organisms. It is also known the vegetation brings organic forms of selenium to the sediment and hence into the diet of higher trophic level organisms.

Selenium chemistry is very complex in sediments and not well understood due to the lack of analytical tools to measure specific chemical forms. The following is an abbreviated list of some of the forms of selenium reported to be present in sediments under reducing conditions: elemental selenium , selenium hydrogen sulfide (SeH_2S), Hydrogen selenide (H_2Se), dimethyl and trimethyl selenide [$(CH_3)_2Se$ & $(CH_3)Se_2$], dimethyl diselenide $(CH_3)_2Se^{-2}$, seleno-diglutathione, seleno-methionine and other organo-forms, copper and several other metal selenides ($CuSe^{-2}$). A critical factor in assessing the selenium chemistry is the eh of the sediment environment. In aerobic sediments, such as the surficial layers in flowing waters or low organic carbon environments, the reduced forms (and organic forms) will not predominate in sediments.

9. Which form (or combination of forms) in sediment are most closely correlated with chronic effects on aquatic life in the field? (In other words, given current or emerging analytical techniques, which forms of selenium in sediments would you measure for correlating exposure with adverse effects in the field?)

   I would measure total selenium; there are no speciated forms of selenium which have been closely correlated with benthic organism or water column effects. Effects that are seen in the field are typically on higher trophic level species such as fish and birds. Effects are not often observed on sediment benthos.

10. In priority order, which sediment quality characteristics (e.g., TOC, etc.) are most important in affecting the chronic toxicity and bioaccumulation of selenium to freshwater aquatic life under environmentally realistic exposure conditions? Of these, which have been (or can be) quantitatively related to selenium chronic toxicity or bioaccumulation in aquatic organisms?

    The list is very short. To date, only total organic carbon (TOC) has been closely correlated with total selenium in the sediment and the potential for chronic effects. These data are limited to flowing waters (lotic systems) and to the Arkansas River system (Van DerVeer and Canton 1997). This research does suggest that there is merit in further understanding the role of carbon in controlling bioavailability or as a surrogate measure for the potential for inorganic forms to be converted to organic forms and hence, a surrogate measure for the potential for chronic toxicity. This is somewhat analogous to carbon normalization for non-polar organics although in this case it does not appear to be due to soption of selenium to carbon in the sediment. Direct correlations of TOC or other sediment quality characteristics with chronic toxicity are lacking. Temperature could be an important factor for Northern aquatic systems where the conversion of inorganic forms to organic forms would be expected to be slower than Southern warm water environments.

11. How certain are we in relating water-column concentrations of selenium to sediment

concentrations? What are the primary sources of uncertainty in this extrapolation?

Once again, the most definitive work in this area has been done by Van DerVeer and Canton (1997) relating total recoverable water-column selenium with total sediment selenium. A correlation coefficient of 0.79 was obtained without sediment carbon correction and with carbon correction a correlation coefficient of 0.93 was obtained. Earlier work done by Lemly related sediment concentrations to observed biological effects in various aquatic systems, but did not correlate sediment selenium with water-column concentrations of selenium.

IV. Cross-Cutting Technical Issues Associated with Chronic Criterion

12. How does time variability in ambient concentrations affect the bioaccumulation of selenium in aquatic food webs and, in particular, how rapidly do residues in fish respond to increases and decreases in water concentrations?

In general, variability in ambient concentrations slowly affects the bioaccumulation of selenium in higher trophic level systems. By slow, I mean over a period of months as opposed to days. The principal reasons for this is due to the fact that there are several rate limiting steps in the conversion of inorganic forms of selenium to organic forms followed by uptake and food-chain transfer of the selenium to higher trophic levels. This approach assumes a dietary pathway. Exceptions to this do occur where there are severely elevated levels of selenium in a given discharge and where the primary route of uptake by the fish is via the gill. In this case, laboratory experiments have shown that the rates of uptake and depuration can be somewhat faster and tissue levels can vary by an order of magnitude over a period of a month in fish. Selenium is an essential element for fish and at low levels of selenium in the water (1 ppb or less) fish (and other aquatic organisms) conserve selenium to meet their needs. Hence, tissue levels remain somewhat constant at environmentally relevant concentrations. Experiments with very low concentrations of selenium (<0.01 ppb) result in very large bioconcentration factors for selenium. In summary, bioconcentration factors for selenium and other essential metals are inversely related to the water concentration.

13. To what extent would the type of ecosystem (e.g., lentic, lotic) affect the chronic toxicity of selenium?

The type of ecosystem and specific ecosystem components appears to play a major role in controlling the potential for chronic toxicity to be expressed in both aquatic and avian species. The work of Van DerVeer and Canton (1997), Canton and Van DerVeer (1997) and Bowie et al (1995) support this conclusion. Lotic systems appear to lack the necessary anaerobic zone(s) and standing vegetation to result in a significant conversion of inorganic forms of selenium to organic forms with subsequent food-chain transfer of the organo-forms to higher trophic levels where embryonic effects are observed. Other factor distinguishing lotic from lentic environments are hydraulic retention time and retention of carbon in the system. Lotic environments have shorter hydraulic retention times and they have much less carbon in the sediments and thereby lack the storage and potential for conversion of inorganic forms to organic forms relative to lentic systems. This is an area where more research is needed. However, there is mounting field collected data supporting the idea that site specific conditions are important in controlling the potential for chronic toxicity to aquatic life from selenium.

Site-specific approaches for determining water quality criteria for aquatic organisms:

Several approaches were considered as to how one might derive a site specific water quality criterion (WQC) for aquatic organisms. First the use of indigenous species and or a water effects ratio (WER) approach were considered. The WER ratio approach does not look promising because selenium bioavailability in the water column is not significantly altered by site conditions such as suspended solids, dissolved organic carbon, hardness or other factors thought to influence the bioavailability of copper, cadmium and other divalent metals (however, this has not been thoroughly investigated). The use of indigenous species probably would not alter the existing WQC because it is based on a single study in Belews Lake and is not derived from the standard EPA Guidelines for WQC. However, exceptions to this conclusion would be ecosystems for which the Belews Lake data (and WQC) are not applicable; for example the Great Salt Lake, Salton Sea or ephemeral streams. These are clear examples where one would question the applicability of existing WQC and the use of indigenous species could be appropriate. Following this line of thinking, one would have to ask how applicable a WQC derived from a Southern lentic lake (Belews Lake) applies to Western/Northern lotic systems?

Second, if sufficient data were available a generic or global model could be developed for the relationship between water-borne selenium and tissue-residue selenium which incorporated a dietary component in the model. This would be patterned after the approach that Skorupa and Ohlendorf (1991) presented for birds. Site -specific data could then be used to determine whether or not site conditions result in significantly more or less accumulation of selenium. The site to global model ratio could be used to provide a site-specific modification of the WQC. This approach can be done, but requires a lot of data.

Third, another alternative which might provide a practical and near-term approach to setting site-specific WQC would be to adopt an approach similar to that developed by Barrick et al. (1988) - Apparent Effect Threshold approach or the Threshold Effects Level approach published by Long and Morgan. This approach to developing a threshold where effects can be seen takes advantage of field data and compares the concentration where effects are never seen versus the concentration where effects are always observed. In between these two values lies the apparent effects threshold. A casual review of the literature for flowing water systems versus lentic systems suggests that there are significant differences in the levels of selenium in the water where effects are observed. Compilation of the existing data should allow for developing lotic and lentic threshold values. Site modifications could potentially be made by adjusting the existing WQC by a factor based on the aforementioned approach to accommodate lotic systems.

Gary Chapman

I will be away from my office all of next week and the following week through May 13. As a result, I have to submit my remarks and recommendations now, without further study of the materials. As an expert on water quality criteria, their site-specific modifications, and criteria guidelines, but with only a nodding acquaintance with past selenium studies, I base my comments regarding selenium primarily upon my reading to date of the materials provided. The main points that I wish to emphasize are in bold, and represent my stronger opinions.

Environmental Compartment. Because of the complex nature of selenium (Se) chemistry and its close relationship to toxicity and bioaccumulation, it is difficult to establish a simple water column concentration criterion. Even in the relatively simple case of mercury, EPA was compelled to establish a criterion that used tissue residues to trigger further site studies of mercury concentrations. In the case of mercury, water concentration exceedence triggers tissue residue investigations targeting FDA action levels. The more complex nature of selenium chemistry probably will require a similar approach with at least some dependency upon the levels of selenium in resident organisms. In addition, Se concentrations below simple detection levels could lead to adverse tissue levels, so that it may be impractical to use water concentration as a trigger for tissue residue investigations. Finally, there is no FDA action level to trigger a residue-based regulation.

Laboratory toxicity studies have provided reasonable estimates for acute toxicity of inorganic forms of selenium as well as selenomethionine. Laboratory studies that included food-chain organisms allowed to accumulate selenium provide additional effect level estimates for chronic toxicity, but even these studies are usually much less complex than the situation in the field.

Considering that, in nature, chronic toxic effects will almost always include a food-chain component, it seems imperative that chronic criteria include consideration of tissue residues in arriving at any chronic water column criterion. EPA's current selenium criterion indirectly utilized this route of uptake by relying upon effect data from the field in establishing a link between water column concentrations and apparently "safe" and toxic conditions. It is well known that the time- course of exposure from various environmental compartments can be very different, with water being the most variable and sediment concentrations being much slower to change. Tissue residues can variably reflect changes in either or both of these sources, as well as spatial variability in water, sediment, and food chain contamination.

I have relied for the following upon the paper by Lemly (1996), describing his evaluation of the hazard quotient (HQ) method for selenium risk assessment as a general basis for my comments. This paper considers eleven sites having various levels of contamination, and the method rates each of five compartments for hazard with a summation of an overall hazard score. I have tended to ignore the bird egg compartment in my comments because they generally appear the same as that for fish eggs, because they may be considered under a separate wildlife criterion, and because of the potential that the site of collection may not represent the site of contamination (depending upon the species involved and its relative migratory habits).

Without consideration of the original studies from which the data were taken, it is difficult to

fully comprehend the significance of the ranges of concentrations indicated for each site. These ranges may reflect variance due to temporal, spatial, or individual organism/sample differences. If we assume that the hazard rating score for each site is a good indication of the hazard involved, **it appears to me that the best correlation between individual compartments and overall hazard score is achieved by the invertebrates and fish egg component**. Indeed, if one considers only these two components, and then takes the higher of the two hazard ratings, they are the same as the overall score for nine of the eleven sites. For the other two sites (Stillwater WMA and La Plata River) either the rating for fish eggs or invertebrates is one category higher (more severe) than the overall rating. **This pair of compartments represents a reasonably good surrogate for the overall score, and is conservative**.

These eleven sites probably do not represent all types of aquatic habitats at risk, so that generalizations that might work with this data set may be inapplicable to other habitats. Never-the-less, criteria are always established using data with various degree of limitation. **Regardless of how well the hazard protocol method appears to work to characterize potential toxicity problems, there are very clear limitations (even with just these eleven cases) with respect to setting a water column criterion.** This is apparent by inspection of the data sets for the three WMAs where tissue residues are high but neither water column nor sediment appear heavily contaminated. The suggestion that this situation is due to very toxic seleno-organic materials is germane, but not particularly instructive with respect to quantification or characterization of the molecule(s) involved. This data does generate considerable prejudice against the Great Lakes approach of treating seleno-organics as toxicologically proportionate to selenate and selenite.

In summary, I believe that it is currently unwise to set any chronic criterion for selenium that does not take into account tissue residue data. It appears that there may be little data upon which to establish dose-response relationships with seleno-organics common to the field. One could assume that all seleno-organics had bioconcentration factors and toxicity similar to selenomethionine, at least in situations where tissue residues are high but selenate, selenite, and total seleno-organics are low. The apparent bioconcentration factors reported by Besser et al. (1993) for selenomethionine at 0.1 ug Se/L for algae (36,300) and daphnids (382,000) would easily account for the range of invertebrate and fish tissue levels reported for the three WMAs from water concentrations of <1 ug Se/L. To a lesser extent, the data of Rosetta and Knight (1995) and Maier and Knight (1993) suggest relatively high selenomethionine bioconcentration for midge and brine fly. Each of these three studies was short-term and might not represent a reasonable steady-state bioaccumulation, especially for longer lived organisms.

The basic problem with applying the tissue residue approach is defining a safe level on the basis of laboratory and field studies. In this regard it would be very instructive to have Lemly discuss the technical basis for his hazard profile categories for macroinvertebrates and fish eggs (from citations in Fig. 1 of his 1995 paper outlining the protocol). I assume that this will be a component of his presentation to the panel; if not, it really must be addressed at the meeting.

Averaging Period. It is generally recognized that the current averaging periods being used by EPA are generally conservative (short) periods intended to apply to a few rapidly toxic chemicals and a few taxa with relatively short life-cycles. The use of toxicokinetics, at least for mortality, is an attractive and reasonably practical approach. In order to implement this approach with any chemical criteria, the data base requirements would be broader than those necessitated by current methods for criteria development. These include more frequent observations of mortality, especially during the first day or two of the test, and consideration of changing sensitivity or modes of action at critical life stages or as exposures lengthen. Another area of data need (generally ignored in current EPA criteria) is the effect of temperature on acute toxicity and toxicokinetics.

It is possible that the toxicokinetic approach will essentially due away with averaging periods, *per se*, and simply declare a particular exposure regime as either acceptable or unacceptable. **Assuming that the exposure period for acute toxicity will be rather short, and the exposure essentially constant, the Mancini model considered recently by EPA can provide reasonable acute toxicity criteria** (Keith Sappington: what is the status of this evaluation?). Regardless of the current status of this approach, its application to selenium is complicated by the several chemical forms that may need to be considered in the model. For this reason alone, **the approach may need to be site-specific**.

Site-Specific Criteria Guidelines. There are two major considerations under this topic. The more common one is how to modify national criteria to apply to a specific location; the less common one, but perhaps more attractive with selenium, is to have no national criterion, or only a narrative criterion or a default criterion, and then establish “site” criteria where problems are expected.

In this regard, it may be possible to classify sites by type. For example, there may be wetland, stream, or lake/pond habitats that have different, but type-specific, exposure patterns regarding chemistry, chemical and hydrological dynamics, and food-chain types. There may be recognizable differences seen with the source of the material (e.g. fly ash, agricultural drain water, normal geological sources). An approach might be generated that would be followed on a regional or a more local basis to establish a criterion for a specific site, for a specific habitat, and/or a specific source. This could be used for problem areas and a generic national criterion apply elsewhere. Obviously such an approach would require state-of-the-art chemical analysis, biocriteria monitoring, and perhaps toxicity tests (at least using ambient water and food organism samples and perhaps using *in situ* exposures).

In practice, site-specific approaches are often considered too data intensive (meaning too resource intensive), too subject to subjective outcomes, and too often only as either the regulator or the regulated using delaying tactics. Also, in practice, initially using less intensive and quicker procedures only delays the activity ultimately necessary for site-specific approaches, because the technical shortcomings of the easy approach only too quickly become obvious and there is a clamor to “fix” the problem. One way around this issue would be to **establish a generic criterion, but establish a concurrent site-specific approach that would almost automatically be used in problem areas.** Although these comments perhaps

appear non-technical (policy), it is important that we determine the best use of today's technical knowledge, and that may include both the best way to set a site-specific criterion where there are known or suspected problems, plus the best way to develop a generic criterion that is relatively risk-free, but, at the same time, generally attainable. To accomplish this requires a reasonable knowledge of selenium sources and ambient levels, not only in water, but in biota (e.g. if we were to recommend that tissue-residues should trigger a requirement for site-specific studies).

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Premeeting Comments

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Preface

Before addressing the specific questions on this topic, and as the only biogeochemist on the panel, I think it appropriate to briefly review what we know about the aquatic cycle of selenium, both in fresh and marine waters (indeed the cycles are essentially the same). Such a review is needed since understanding the cycle allows one to rationally evaluate toxicological data and decisions. In the water column selenium can exist as the dissolved inorganic ions selenate (SeVI) and selenite (SeIV), as dissolved organic selenide in which Se-II is covalently bonded to carbon moieties, and as particulate selenium (i.e., in "suspended particulate matter") that includes organic selenide, adsorbed selenite + selenate, and elemental selenium (Se0). In uncontaminated, fresh waters, dissolved organic selenide and selenate are usually the predominant forms (i.e., nearly equimolar), with selenite being a minor species (see Cutter, 1989; Cutter, 1991; Cutter and San Diego-McGlone, 1990). The chemical "identity" of organic selenide is very relevant to bioavailability questions, but elusive. It is definitely not the free seleno-amino acids (seleno-methionine or -cysteine; Cutter, 1982; Cutter, 1991), but it probably is these seleno-amino acids bound in soluble peptides/proteins (Cutter, 1982; Cutter, 1991; Cutter and Cutter, 1995). In contaminated fresh waters, particularly those associated with fossil fuel combustion or refining, selenite tends to be the predominant dissolved form of selenium (Cutter, 1989; Cutter, 1991; Cutter and San Diego-McGlone, 1990), although in systems such as the San Luis Drain/Kesterson Reservoir in the Central Valley of California selenate was the predominant form (Cooke and Bruland, 1987; Cutter, 1989). This then leads into the biogeochemical cycle of selenium in the water column and the interconversions of dissolved selenium species that I believe makes it very difficult to set criteria based on water

column concentrations or speciation.

In the water column, removal of dissolved selenium is driven by phytoplankton uptake, which is selective, with the rate of selenite uptake being greater than that for selenate or organic selenide. With respect to the latter, the work of Gobler et al. (1997) is the only definitive study of which I am aware that follows uptake of more “realistic” organic selenide (derived from the lysis of phytoplankton) rather than seleno-methionine; it’s uptake by a marine diatom is far slower than that for selenite. In spite of the original dissolved oxidation state, the selenium taken up by phytoplankton is reductively incorporated to organic selenide, primarily seleno amino acids in proteins (e.g., Wrench, 1978; Cutter, 1985; Cutter and Bruland, 1984). While it is possible that selenite or selenate are adsorbed to phytoplankton tissues, solid phase speciation data do not indicate that this is a significant fraction (i.e., > 20% of the total; Cutter, 1985; Cutter and Bruland, 1984; Cutter, 1991). Thus, selenium is transferred to the next trophic level largely as organic selenide (Reinfelder and Fisher, 1991) in spite of the original water column speciation, and this appears to hold with further steps in the food web (Fisher and Reinfelder, 1995). The regeneration of particulate selenium in detritus to the dissolved state is a multi step process where particulate organic selenide is regenerated during bacterial respiration/degradation of organic matter to dissolved organic selenide, which then oxidizes to selenite and then to selenate; the rate of the latter step is extremely slow, accounting for the persistence of thermodynamically unstable selenite in all natural waters (Cutter, 1982; Cutter and Bruland, 1984; Cutter, 1991; Cutter, 1992). The net result is that any form of selenium can be taken up, but at different rates, and it will be regenerated to the biologically-preferred form, selenite, during the cycle.

This cycle can be put into an entire ecosystem context as depicted in the attached figure. This figure is for an estuary and is the model currently under investigation by our lab, and those of Nick Fisher (SUNY, Stony Brook), Sam Luoma (USGS, Menlo Park), and David Hinton (UC Davis) with funding from NSF and the State of California. I’d suggest that it is just as viable in rivers and streams, where the horizontal advective/diffusive transport terms are large and unidirectional, or in lakes (horizontal transport minimal). In this model, biotic uptake from the dissolved state (SeIV, SeVI, or org Se-II) is accomplished by the primary producers,

and the resulting organic selenide (or adsorbed SeIV+VI) are transferred to the next trophic levels ("consumer organisms"). However, some consumer organisms, in particular for this model, bivalves, can also take up selenium from sediments, which, in addition to organic selenide and adsorbed SeIV+VI (largely from detritus, but also produced *in situ*), contain elemental Se from the dissimilatory reduction of selenite and selenate (Oremland et al., 1989; Velinsky and Cutter, 1991; Cutter, 1992). This is another biotic uptake vector and there is some effect by solid phase speciation on selenium's assimilation into bivalves (organic Se-II>Se0; Luoma et al., 1992), but the selenium still resides in organic matter after uptake. For trophic transfer, the work of Fisher and his colleagues (see Fisher and Reinfelder, 1995 for a review) shows that the primary control on selenium's trophic transfer (as measured by assimilation efficiency) is really its phase speciation...whether it is in a cell's cytosol or tissues. Thus, the important parameters influencing the concentration of selenium in a higher organism are: selenium speciation in the water column and sediments, rates of uptake of the various selenium forms by primary producers (and consumers) relative to the rates of physical transfer in the system (i.e., residence times), and the assimilation efficiency and net rate of trophic transfer (uptake - depuration) between the various trophic levels. It is a whole ecosystem problem that requires quantitative information on the physical setting (advection/diffusion), the biogeochemical cycle controlling the water column and sediment speciation (dynamics between primary producers and the microbial loop; aquatic chemistry of selenium), and the trophic structure/interactions of the ecosystem.

One other important topic concerning any criteria for selenium is its analytical chemistry. Because kinetics play such a dominant role in the environmental behavior of this element (Cutter, 1992), one should avoid assumptions and instead make empirical observations. This requires analytical methods that are precise (random errors in the analysis have to be far less than the environmental variability), accurate (minimize systematic errors; are you measuring the chemical form you think you are?), and able to determine speciation in both the dissolved and particulate phases. Related to this, when examining literature on uptake, etc. one must question whether the selenium form added was correct (e.g., selenite contamination is pervasive in selenate standards) and remained in that form during the

experiment (i.e., the recycling of selenium changes its chemical speciation). Furthermore, radiotracers only tell us that it is selenium, but not what chemical form it is.

Water Column-Based Chronic Criterion

1. As noted in the review above, all forms of dissolved selenium are important since they all can be taken up (they are all bioavailable), albeit at different rates, and then recycled to other forms.
2. I do not believe that a legitimate correlation between dissolved form and chronic effects can be made. Perhaps the best examples are Kesterson Reservoir where selenate (and organic selenide in the last ponds in the flow scheme) predominated and waterfowl mortality was clearly a problem, and the power plant cooling reservoirs in North Carolina, Hyco (Carolina Power and Light) and Belews (Duke Power), where selenite predominated and complete collapses of their fisheries occurred. In terms of measurements, it is essential to separate dissolved and particulate forms of selenium via filtration, and each should be determined uniquely, not by difference (i.e., filter the sample through a 0.2 µm membrane filter, determine dissolved selenium speciation in the filtrate, and determine selenium speciation on the filtered material). The key here is dissolved selenium is that fraction available to primary producers, while particulate selenium (which includes the primary producers in the water column) is that available for trophic transfer to higher organisms.
3. I can find no compelling reason to rank any water quality parameter as being important to selenium's bioavailability without resorting to site-specific cases. In the case of sulfate, if sulfate inhibits dissolved selenate uptake, then oceanic phytoplankton which live in waters with 26 mmol/L sulfate and less than 0.4 nmol/L selenate (8 orders of magnitude difference) would never take up selenate, but they do. A similar situation occurred in Kesterson Reservoir. Riedel and Sanders (1996) suggest that phosphate may be important for one green alga, but it would be difficult to separate this effect from simple growth stimulation.

Tissue-Based Chronic Criterion

4-5. As noted in my review, essentially all tissue selenium is in proteins as selenide. Thus, there is little to differentiate.
6. There may be subtle differences in the types of tissues/location of selenium (so called "phase speciation") that effects selenium's assimilation efficiency and trophic transfer. Again, Reinfelder and Fisher (1991) found selenium in soluble peptides of the cytosol more bioavailable.
7. As noted in my review above, the concentration of selenium in tissues of higher organisms such as fish may be correlated with water column concentrations, but there are many factors (rate constants, assimilation efficiencies) that would create considerable error in the estimate (I say this even though I have been involved in such estimates and co-authored papers on simulation models that seek to make these calculations).

Sediment-Based Chronic Criterion

8-9. Again referring to the review above, all forms of selenium are found in sediments, although the primary ones are organic selenide (most bioavailable for trophic transfer) and elemental selenium (less available), depending on the redox state of the sediment and overlying water (see Cutter, 1991). There have been no definitive studies of toxicity and sedimentary selenium speciation to my knowledge, with the work of Luoma et al. (1992) being the best in terms of analytical methods (accuracy of speciation) and environmental relevance (concentrations, speciation, organisms).
10. Like the water column, it is probably unwise to seek pseudo correlations/factors influencing selenium's bioavailability. There may also be some irrelevant correlations since TOC will drive sediment anoxia which preserves sedimentary selenium (Velinsky and Cutter, 1991; Cutter, 1992), making high Se correlate with high TOC.
11. By knowing the residence time of water in a system, the concentration of sedimentary selenium can be accurately calculated from the water column concentrations (see Cutter, 1991 for an example). This is derived from mass balance calculations, where selenium introduced to a lake either is removed to the sediments via biotic uptake/detrital flux or by precipitation of

Se(0) during hypolimnetic anoxia, or lost by volatilization (minor), or removed by water outflow (i.e., relative rates of water removal vs. removal to the sediments). Thus, the longer the water residence time (less outflow), the more is found in the sediments. The primary errors here are in the rate constants for removal and sediment regeneration.

Cross-Cutting Issues

12. Time variability is a crucial factor, and as noted in the sediment comment above, it is a relative rate problem. Using primary producers as an example, if the water column concentrations of selenium vary faster than the uptake rates, then the concentration of organic selenide in the phytoplankton will be controlled by the average water column concentration, whereas slow water column variations will make the phytoplankton concentrations represent the instantaneous concentrations. This effect will be diminished with each step in the food web, but other temporal variations will be then included. Overall, at the top of the food chain, fish should average out the water column behavior of selenium.

13. The type of ecosystem/food web will strongly control the effects of selenium. In a lentic system, more selenium can accumulate in the sediments, affecting any benthic food web, and relatively slow rates of reactions will become more important (e.g., the slow rate of selenate uptake by phytoplankton will manifest itself, whereas in a lotic system it would be inconsequential). As a result, the modeling approaches to simulating these systems, and therefore predicting selenium concentrations and bio-effects in the upper trophic levels, would be substantially different.

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I. Technical Issues Associated with a Water-Column-Based Chronic Criterion

Selenate (Se^{+6}) and selenite (Se^{+4}) are the two most common forms of selenium in the water column, with Se^{+6} more prevalent under strongly oxidizing conditions of fast moving waters and Se^{+4} likely to be more prevalent in lentic systems. Organic forms of selenium (e.g., the biological forms of seleno-DL-methionine and seleno-DL-cysteine or selenocystine) are toxicologically more important than the inorganic forms, but generally are found at very low concentrations in water (see Bowie et al 1996 or Scott 1991). Elemental selenium is very insoluble and selenide (Se^{-2}) generally cannot exist in the natural aqueous environment because reducing conditions cannot be met (Scott 1991).

The relative toxicological importance of selenate and selenite may vary by species, although selenite appears to generally be more toxic to aquatic organisms than selenate. Selenite is 2-4 times more toxic to *Hyallela azteca* than is selenate (Brasher and Ogle 1993). Similarly, selenite is more toxic than selenate to chinook salmon *(Oncorhynchus tshawytscha*) and coho salmon (*O. kisutch*) in short-term (acute) studies (Hamilton and Buhl, 1990). However, *Chironomus decorus* larvae are more sensitive to selenate, then selenite in a water-only exposure (Maier and Knight 1993), although the water column is not likely to be the primary exposure medium for benthic invertebrates or fish (food selenium is a more important exposure route). The acute toxicity of the two forms of selenium appears to be additive, but this is not known for chronic exposures. Therefore, until such studies are performed with chronic toxicity studies I would recommend that both selenate and selenite be measured in the water column, rather than assuming additivity and measuring only total selenium.

The following water quality characteristics, listed in order of priority, are most important in affecting the chronic toxicity and bioaccumulation of selenium to freshwater aquatic life: dissolved oxygen (DO), Eh, pH, sulfate, TOC, iron, other metals (especially mercury and copper).

Toxicity relationships derived from acute toxicity and water quality characteristics are not very applicable to chronic toxicity situations in the field for organisms other than phytoplankton and bacteria. Only the primary producers are affected mainly by water-borne selenium; all other organisms receive most of their exposure from food or sediment (e.g., Bowie *et al.* 1996). Therefore, acute toxicity studies that look at water-only exposures are likely to underestimate chronic toxicity to benthos and fish.

In addition, uptake of selenium by benthic macroinvertebrates is not very predictable from water-only selenium concentrations (Adams *et al.* 1998). This is due to differences in selenium species in the water (generally reported as total selenium), differences in water and sediment physical parameters, whether the system is oxic or anoxic, and volatilization rates of selenium, that may be as high as 25-35% in shallow systems (Hanarm *et al.* 1996).

II. Technical Issues Associated with a Tissue-Based Chronic Criterion

Within organisms, the most toxicologically important forms of selenium are the amino acid-substituted forms, selenomethionine, selenocysteine, and selenocystine. Selenium causes adverse biological effects by substituting for sulfur in these amino acids and, subsequently, in various protein enzymes such as glutothione peroxidase (GPx). The loss of function of GPx results in a decreased antioxidant ability and subsequent destabilization of cell membranes. Selenium also interfers with cell division, thereby causing the terata associated with selenium-induced reproductive disorders. The inorganic forms of selenium must be converted to these organic forms prior to exerting their effects. However, nearly all studies that correlate tissue selenium concentrations with either biological effects or with water or food concentrations have measured only total selenium. Even this measure has a relatively reasonable correlation with effects (see below).

Tissues best correlated with overall chronic toxicological effects thresholds for selenium in aquatic organisms are gravid fish ovaries and whole body concentrations in benthic invertebrates (Lemly 1996). Whole body residues in fish are not correlated with incidence of effects if all species are considered together; however, by considering each species separately, a reasonable fit of an exponential function is expressed (Lemly 1993). Muscle tissue plugs (or fillets) also have been correlated with

effects. Note that all tissue concentrations should be expressed on a dry-weight basis when making these types of correlations.

We are not very certain in relating water-column concentrations of selenium to tissue-residue concentrations in top trophic-level organisms such as fish. This is because fish receive their primary exposure through food (i.e., greater amounts of selenium but also the selenium is in the more toxic form of amino-acid substituted selenides) not from the water. The amount of exposure from food is dependent upon the physical conditions of the water column (see above) as well as the sediments (see below). Moreover, the types of fish prey items present will affect how much selenium has been accumulated into the fish diet. For example, the organoselenium forms are accumulated more rapidly by benthic invertebrates than are the two inorganic forms (Maier and Knight 1993; Rosetta and Knight 1995; Reidel *et al.*, 1991). Both selenomethionine and selenate uptake from water by phytoplankton seem to be active physiological processes, whereas selenite is merely mechanical binding kinetics although over the long term uptake rates of the two inorganic forms are similar (Bowie et al 1996). For bacteria, uptake of selenite is more rapid, even over the long term, than is selenate (Bowie et al 1996).

Therefore, in order to use tissue-to-effect correlations for setting a water-based criterion, we need to be able to predict what water concentrations will result in tissue concentrations that are known to be correlated to adverse effects. This means first understanding what food concentrations (i.e., invertebrate tissue concentrations) cause effects in fish – this we can do with a fair amount of success. The next step is to predict what sediment or phytoplankton concentrations result in the various tissue concentrations in invertebrates. This we can do with lesser success. For sediment – benthos relationships, the variability in sediment selenium species and benthic community composition confound the relationship (see below). The phytoplankton – benthos relationship has not been measured. Direct correlations of water – benthos concentrations are not very good (Adams *et al*. 1998), so the final step is correlating water concentrations with sediments or phytoplankton. This we can do with some degree of certainty, at least for a few systems. Bowie *et al*. (1996) and Van Derveer and Conton (1997) have attempted to mathematically model these relationships and have been fairly successful for particular systems. The challenge, now, is to see how this model can be parameterized for a larger variety of systems.

III. Technical Issues Associated with a Sediment-Based Chronic Criterion

The dominant selenium species in sediments are elemental selenium and organic selenium (Van Derveer and Canton 1997), although this likely is dependent upon the type of sediment (oxic versus anoxic).

For sediments, measurement of total selenium may be sufficient, without the need to speciate the types of selenium present (Van Derveer and Canton 1997). However, this probably needs to be investigated further with particular emphasis on comparing lotic versus lentic systems.

The following sediment characteristics, listed in order of priority, are most important in affecting the chronic toxicity and bioaccumulation of selenium to freshwater aquatic life: TOC, texture, Eh, pH, sulfate, iron.

Of these, total organic carbon (TOC) is by far the most important factor, with texture and Eh close behind. Van Derveer and Canton (1997) have developed an empirical model of water-to-sediment selenium transfer in lotic systems (western streams) that predicts sediment accumulation as a function of dissolved selenium in the water column and sediment TOC.

IV. Cross-Cutting Technical Issues Associated with Chronic Criterion

Fish tissue residues respond relatively slowly to changes in water selenium concentrations (Lemly 1996). This is partly because fish are exposed primarily through their food source (benthic invertebrates) which, in turn, are exposed through sediments and through phytoplankton which take up selenium from the water column. Phytoplankton and bacteria accumulate selenium rapidly, reaching maximum levels within 5 to 6 days or less (Sanders and Gilmour 1994). If the assumption were made that depuration rates are no greater than uptake rates, turnover time for these organisms would be about two weeks. Benthos and fish respond much more slowly to changes in water column selenium due to the retention of selenium in sediments (Bowie *et al.* 1996). The rate of loss of selenium from sediments depends upon volatilization rates (from 5 – 35%, depending upon the type of bacteria present which are a function of the redox potential of the sediments), the amount of binding to sulfides and iron, and the reducing environment of the sediments (and consequent sequestration as elemental or reduced forms of selenium). Half-lives of sediment selenium also depend upon the species of selenium. For example, in microcosm studies, half-lives were measured at 7 days for selenomethionine, 20 days

for selenite, and 33 days for selenate (Besser *et al.* 1989). While these relative relationships probably hold for field systems, it is unlikely that the absolute numbers would be the same.

The type of ecosystem (lentic or lotic) will have a large influence on the chronic toxicity of selenium to aquatic organisms. This is due to the differences in speciation of selenium in the water column and, more importantly, in the sediments as a result of different amounts of oxygen. In addition, different types of bacteria and phytoplankton will be present. Since these organisms are responsible for the majority of the bioaccumulation of selenium into the food web (with BCFs up to 3 orders of magnitude), as well as for the amount of methylation and subsequent volatilization out of the system, they will have a large influence on the flux of selenium through the aquatic biota. Different species take up the various forms of selenium at different rates and to different concentrations. It is likely that lotic systems can have higher water concentrations of selenium than can lentic systems, prior to causing overt effects to fish. This has not be systematically studied, however, although the system model of Bowie *et al.* (1996) might be useful for examining such predictions and identifying key relationships in more detail.

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COMMENTS TO TECHNICAL CHARGE ASSOCIATED WITH EPA Se WORKSHOP

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I. Technical Issues Associated with a Water-Column-Based Chronic Criterion

1. Besides selenite and selenate, which other forms of selenium in water are toxicologically important with respect to causing adverse effects on freshwater aquatic organisms under environmentally realistic conditions?

It appears that dissolved organoselenium form(s) including those in dissolved organic matter may be very important in highly productive systems in terms of foodchain transfer and toxicological relevance. However, there is little information regarding the chemical nature of these forms. Based on known stability of different organoselenium compounds, selenomethionine may be one of the more persistent forms that should be investigated.

In addition to dissolved form(s), particulate Se form(s) derived from planktonic organisms and detritus are also important to investigate because these forms are ingested by aquatic invertebrates and fish, which represent a major route for Se bioaccumulation and transfer through the foodcahin (e.g. Hermanutz et al., 1992; Wang et al., 1996). There is also evidence that particulate Se responds to changes in waterborne Se concentration rapidly (Bowie et al., 1996). As such, it may be a good short-term integrative indicator of waterborne Se status.

2. Which form (or combination of forms) of selenium in water are most closely correlated with chronic effects on aquatic life in the field? (In other words, given current or emerging analytical techniques, which forms of selenium in tissues would you measure for correlating exposure with adverse effects in the field?)

Note: Your response should include consideration of operationally defined measurements of selenium (e.g. dissolved and total recoverable selenium), in addition to individual selenium species.

The literature data on this issue is very limited. However, I believe proteinaceous Se form(s) both as total and as specific form(s) such as selenomethionine warrant measurements for the reasons given in Point #4. In addition, the analysis for these forms is now practical to perform.

3. A) in priority order, which water quality characteristics (e.g. pH, TOC, sulfate, interactions with other metals such as mercury) are most important in affecting the chronic toxicity and bioaccumulation of selenium to freshwater aquatic life under environmentally realistic exposure conditions?

I could not rank these characteristics based on available literature data but it is clear that pH (e.g. Riedel and Sanders, 1996), TOC, salinity (particularly sulfate salinity, e.g. Riedel and Sanders, 1996; Ogle and Knight, 1996), temperature (e.g. Lemly, 1993), and presence of other metal/metalloid ions such as mercury, cadmium, copper, arsenic, and molybdenum (e.g. Naddy et al., 1995) would have significant effects on Se impact on aquatic life. The importance of each characteristics will depend on the site conditions, foodweb, and biogeochemistry. Some of these effects appear to be antagonistic and some are synergistic, depending on a number of factors (e.g. category of organisms, life stages of organisms, concentration range, specific forms, etc.)

B) Of these, which have been (or can be) quantitatively related to selenium chronic toxicity or bioaccumulation in aquatic organisms? How strong and robust are these relationships?

Temperature have been related to fish chronic toxicity (Lemly, 1993) while sulfate, phosphate, and pH have been related to Se bioaccumulation in phytoplankton (Riedel and Sanders, 1996). There have been other studies of the influence of sulfate on Se bioaccumulation but they were conducted as acute toxicity testing. Since the temperature, sulfate, and pH effect was demonstrated on only a few species, it is difficult to conclude whether the observed relationships are generally applicable.

C) How certain are applications of toxicity relationships derived from acute toxicity and water quality characteristics to chronic toxicity situations in the field?

The acute toxicity tests for Se are generally conducted using water exposure at environmentally unrealistic concentrations and in very short time periods. There is a general consensus from the literature that Se exposure to aquatic consumers is mainly mediated through diet and that reproductive failure is a key toxic effect resulting from Se exposure. Neither is addressed with the established acute toxicity assay. In addition, there are indications that Se bioaccumulation, biotransformation pathways, and therefore toxic actions depend on exposure concentrations and length of exposure, and that extraploating effects expressed at high/short to low/long exposure would be difficult and complicated. For example, both bioconcentration factor and toxicity (e.g. LC50) of Se depend on Se exposure concentration and length (e.g. Brasher and Ogle, 1993; Besser et al., 1993); this dependence does not appear to be easily defined. Biotransformation pathways in microorganisms change with Se treatment concentrations (e.g. Fan et al., 1997; Fan et al., submitted to ES&T). In particular, the extent of Se incorporation into proteins and proteinaceous Se forms are

dependent on Se treatment concentrations, which may have important implications in regard to adverse effects (see also Point 4).

II. Technical Issues Associated with a Tissue-Based Chronic Criterion

4. Which foms of selenium in tissues are toxicologically important with respect to causing adverse effects on freshwater aquatic organisms under environmentally realistic conditions and why?

Total tissue Se burden does not appear to be a reliable indicator of adverse effect since there are many cases where normal and adversely affected organisms have comparable Se burden (e.g. the normal and deformed fish from Belews Lake had equivalent whole body Se concentrations, Lemly, 1993). Tissue burden of Se may not be indicative of foodchain transfer and bioaccumulation potential either since Se retention by a given organisms depends on many factors such as predator/prey relationship, ingestion rate, assimilation efficiency, depuration rate, Se forms, and environmental conditions (e.g. Wang et al., 1996; Saiki et al., 1991.). Moreover, tissue burden does not explain the differential sensitivity of aquatic organisms to Se exposure.

No direct information is available regarding the form(s) of Se in tissues that are toxicologically relevant in aquatic systems. However, there are hints from the literature that selenomethionine may be a key. Selenomethionine is generally bioconcentrated to a greater extent and more toxic to aquatic life than selenite and selenate (e.g. Besser et al., 1993; Woock et al., 1987; Ingersoll et al., 1990). Dietary selenomethionine toxicity in laboratory trials closely approximates that of field-collected organisms (e.g. Hamilton et al., 1990; Heinz et al., 1996; Woock et al., 1987). On the other hand, it is unclear whether FREE selenomethionine in tissue is relevant since none of these studies measured the free selenomethionine concentration in tissues. In the tissues that we have surveyed (micralgae, vascular plants, mushroom, and bird blood), there is very little free selenomethionine. It is also difficult to rationalize selenomethionine toxicity because its selenol group is blocked by methylation.

Indeed, selenomethionine does not catalyze the production of superoxide anion and its in vivo toxicity is low compared with other Se compounds that catalyze this reaction (Spallholz, 1998)

It is more likely that selenomethionine in tissue proteins (proteinaceous Se-Met) may be most toxicologically relevant based on the following observations. Free selenomethionine

concentration in algae isolated from agricultural drainwaters is very low (e.g. Fan et al., 1997), while proteinaceous Se-Met is a significant fraction of biomass Se in these algae and aquatic birds (Fan et al., submitted to ES&T & unpublished results). Although free Se-Met has been reported in some other algae (e.g. Bottino et al., 1984), the identity of Se-Met has not been structurally confirmed in these studies. This is a problem since we could not find GC-MS evidence of Se-Met in an HPLC peak assumed to be Se-Met based on retention time.

There are also indirect evidence that Se present in the protein fraction of aquatic organisms is very relevant to Se bioaccumulation and adverse effect. Bioconcentration factors of Se in aquatic invertebrates decrease with increasing Se dose from their algal diet (e.g. Besser et al., 1993; Knight, 1988). This is consistent with a decreasing allocation of Se and Se-Met into algal proteins with increasing algal Se burden (Fan et al., submitted to ES&T). A Chlorella agla with a much lower % Se allocation into proteins exhibited a much higher tolerance towards Se treatment than a filamentous cyanophyte (Fan et al., in press; Fan et al., submitted to ES&T). Most Se in fish tissues is associated with protein-rich organs (Lemly, 1993) and aquatic microorganisms contain significant amounts of selenoproteins (Wrench, 1978; Weiss et al., 1965). Se depuration from tissues is consistent with a two-compartment model (Wang et al., 1996); the less depuratable compartment may represent the protein compartment. Regardless of the forms of Se fed to aquatic algae or invertebrates, the tissue Se burden in their consumers such as fish exhibit a linear relationship with the food-borne Se dose, which suggests conversion to a common metabolic pool by the diet organisms (Besser et al., 1993), such as proteins. Acute toxicity to aquatic invertebrates and fish increases with length of exposure (Brasher and Ogle, 1993; Hamilton and Buhl, 1990) regardless of the forms of Se exposure and that different Se forms appear to have a common mode of toxic action in fish (Hamilton and Buhl, 1990). Based on known Se biochemistry, this toxic action may be related to proteinaceous Se.

5. Which form (or combination of forms) of selenium in tissues are most closely correlated with chronic effects on aquatic life in the field? (In other words, given current or emerging analytical techniques, which forms of selenium in tissues would you measure for correlating exposure with adverse effects in the field?)

Proteinacious Se and proteinacious selenomethionine, in particular, should be relevant and practical to measure (see Point 4). Other selenocompounds with free selenol and/or diselenide groups may also be important, but they are unstable to extraction and their analysis is more difficult at the present time.

6. Which tissues (and in which species of aquatic organisms) are best correlated with overall chronic toxicological effect thresholds for selenium?

Since reproductive disturbance of higher trophic organisms is a key toxic expression of chronic Se exposure, fish and bird reproductive organs and eggs should be a good choice (Hermanutz et al., 1992). However, in case of difficulty in obtaining these tissues, blood samples collected during reproductive season may be a good surrogate.

7. How certain are we in relating water-column concentrations of selenium to tissue-residue concentrations in top trophic-level organisms such as fish? What are the primary sources of uncertainty in this extrapolation?

I don't think we can be certain in relating waterborne Se concentrations to tissue residue concentrations in fish because there are cases where Se concentrations in fish eggs do not correlate with waterborne Se concentrations based on the data compiled by Lemly, 1996. These deviations are to be expected since the pathway from water through foodweb could vary among different aquatic ecosystems.

III. Technical Issues Associated with a Sediment-Based Chronic Criterion

8. Which forms of selenium in sediments are toxicologically important with respect to causing adverse effects on freshwater organisms under environmentally realistic conditions?

Again, the literature information regarding this issue is very limited. However, it can be reasoned that Se forms in detritus and possibly sediment organic matter would be toxicologically important since there is evidence that Se is highly concentrated in detritus (e.g. Saiki and Lowe, 1987) and that these are important food source for benthic organisms.

9. Which form (or combination of forms) in sediment are most closely correlated with chronic effects on aquatic life in the field? (In other words, given current or emerging analytical techniques, which forms of selenium in sediment would you measure for correlating exposure with adverse effects in the field?)

Proteinaceous Se and selenomethionine in benthic organisms, detritus, and sediment organic matter would stand a good chance in correlating with chronic effects on aquatic life.

10. In priority order, which sediment quality characteristics (e.g. TOC, etc.) are most important in affecting the chronic toxicity and bioaccumulation of selenium to freshwater aquatic life under environmentally realistic exposure conditions? Of these, which have been (or can be) quantitatively related to selenium chronic toxicity or bioaccumulation in aquatic organisms?

Organoselenium forms in benthic organisms and sediment > total Se in benthic organisms and sediment organic matter > total sediment Se > TOC. Total sediment Se have been related to effects on fish and birds (e.g. Van Derveer and Canton, 1997). However, this correlation is

only tentative since there are uncertainty associated with the sediment Se data and the effect assessment. I should also point out that sediment Se distribution and possibly TOC can be highly heterogenous, varying with location and depth. This issue would need to be resolved before these parameter can be used reliably.

11. How certain are we in relating water-column concentrations of selenium to sediment concentrations? What are the sources of uncertainly in this extrapolation?

I don't think we can relate waterborne to sediment Se concentrations due to a highly variable relationship observed in the field (e.g. Saiki and Lowe, 1987). For example, the waterborne Se concentration could differ by an order of magnitude in two of the California's agricultural drainage ponds, and yet the sediment Se concentrations for the two ponds are comparable. The sources of uncertainty could be sediment characteristics in terms of Se sorption, particulate deposition from water column, Se speciation in water and sediment, algal and microbial community and activity, etc.

IV. Cross-Cutting Technical Issues Associated with Chronic Criterion

12. How does time variability in ambient concentrations affect the bioaccumulation of selenium in aquatic food webs, in particular, how rapidly do residues in fish respond to increases and decreases in water concentrations?

It appears that Se residues in fish do not respond rapidly to changes in ambient concentrations as examplified by the cases of Belews Lake and Hyco Lake (Lemly, 1993; Bowie et al., 1996). Se status in plantonic organisms including microalgae, bacteria, and protozoans tends to be more reflective of ambient concentrations.

13. To what extent would the type of ecosystem (e.g. lentic, lotic) affect the chronic toxicity of selenium?

Se biogeochemistry and foodweb differ among ecosystems, particuarly between lentic and lotic systems. As such, the chronic toxicity of Se is expected to vary with ecosystem type. For example, primary productivity is generally much lower in fast-flowing river than reservoirs and lakes with long residence time. This difference would greatly influence Se biotransformation and bioaccumulation through the foodchain, and therefore chronic toxicity. Other factors such as sulfate could alter the benthic microbial community (e.g. dominance of sulfate reducers), which in turn would affect Se transformations in the sediment. However, it is difficult to quantitatively assess these effects based on existing literature information.

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Technical Charge To Experts

I. Technical Issues Associated with a Water-Column-Based Criterion.

1. Organic forms of selenium such as selenomethionine and selenocysteine may be important to consider in evaluating the toxicological effects in water, and through the food chain. Several studies have shown these organic forms are readily taken up in the food chain and bioaccumulated to concentrations toxic to higher trophic levels (Besser et al. 1993). Several investigators have shown selenomethionine was the most toxic of the two most commonly tested forms to cyanobacteria (Kiffney and Knight 1990), aquatic invertebrates (Maier et al. 1993), and fish (Niimi and LaHam 1976). Maier et al. (1993) cited three papers that demonstrated that upto 60% of the total selenium in aquatic ecosystems may be in the organic form (Robberecht and Van Griekert 1982, Takayanagi and Wong 1985, Cooke and Bruland 1987).

   Nevertheless, the practicality of measuring organic forms of selenium as part of a criterion would be difficult due to the lack of a relatively accessible method for widespread use.

2. Most studies do not speciate selenium into its forms as part their analytical measurement. Some work has been done to differentiate dissolved versus total (particulate) selenium. Graham et al. (1992) compared the cycling of selenomethionine and selenite in a freshwater experimental pond and showed that little difference between filtered (0.45 μm) and unfiltered water samples using radio-labelled compounds.

   Part of the problem in measuring organic selenium compounds in water is that they can be taken up by biota at a rate an order of magnitude faster than selenite (Graham et al. 1992).

3A. My priority order would be 1) interaction with other elements, 2) total organic carbon (TOC), 3) sulfate, 4) pH. I am not aware of any affects on selenium toxicity due to pH. Sulfate does not seem important because in high sulfate aquatic environments, selenium effects do not seem to be ameliorated (Skorupa 1998). Skorupa (1998, p. 345) gives several examples of low and high sulfate aquatic environments where selenium toxicity to biota was not confounded by the presence of sulfate at high concentrations.

TOC has been identified as a component relatively closely associated with selenium concentrations (Stephens et al. 1992 [pages 96-99], Peltz and Waddell 1991) reported a positive relation between concentrations of selenium in pond sediments at Ouray national Wildlife Refuge (NWR), Utah, and concentrations of organic material in sediment.

3B.

3C.

4. Selenomethionine seems to be the most toxicologically important form in tissues followed by selenite, and selenate. However, because of the rapidity that selenomethionine is removed from the water by biota and sediments, it would be difficult to quantify it in water in a meaningful manner. Once selenium is incorporated into the tissues of biota, it's many potential forms would make it difficult to measure (see response to item 5 below).

5. The form of selenium in tissues related to toxic effects is apparently organic in nature based on the work of Stadtman (1974, 1980) who identified selenoproteins in microorganisms and demonstrated that selenium is essential for certain enzymes. If present at greater than essential amounts, selenium saturates the sulfur metabolic pathways, forming deleterious amounts of selenorganic compounds, which can form selenoproteins, but may disrupt whole-animal functions such as growth, behavior, and eventually survival. Stadtman (1974) stated "Because of the greater reactivity and lower stability of selenium compounds compared to the corresponding sulfur compounds, the cell may encounter metabolic problems which eventually can lead to death of the organism."

   For tissue, the most useful approach would be to measure total selenium because of the multitude of organoselen forms that could be present. Several investigators have identified a variety selenium containing amino acids and proteins (Bottino et al. 1984, Stadtman 1980, Wrench 1978). Consequently, trying to quantify selenium residues in tissue based on one form or another would not fruitful.

6. Most of the literature is based on whole-body residues. There is limited information on concentrations in various tissues including gills, skin, liver kidney, spleen, heart, muscle, eggs, blood, plasma, and other tissues. Because he majority of information is in whole-body tissues, I recommend that whole-body residues should be the basis for a tissue-based criterion. Some of the other tissues such as liver, kidney, and eggs may have higher residues of selenium, but the small amount of tissue available from small fish would make analysis impractical. Composites of whole-body would be necessary for aquatic and benthic invertebrates and plants.

   A tissue-based criterion for selenium would be better than an water or sediment-based criterion because tissues integrate all exposures and results in the biological effect that criterion are established to protect, i.e., aquatic life. Several studies have documented adverse effects in aquatic life and linked adverse effects with either waterborne, dietary, or combined water and dietary exposure to selenium (Table 1).

Examining these studies shows a convergence of whole-body residues in the 4-5 μg/g dry weight range, regardless of exposure route. In general, background selenium residues in fish typically fall in the <2 μg/g range, with a few exceptions.

Table 1. Selenium concentrations in young fish exposed to selenium in the diet or water and adverse effects observed.

Exposure route, species, weight (g), age	Se form	Exp. period (day)	Treated: Se exp. conc. (μg/g; μg/L)	Treated: W-B Se (μg/g)	Effect	Control/Reference: Se exp. conc. (μg/g; μg/L)	Control/Reference: W-B Se (μg/g)	Ref.[1]
Diet								
Rainbow trout								
79 (NG)	Selenite[2]	294	9	NG[3]	Mortality	NG	NG	1
1.3 (NG)	Selenite[4]	140	13	5.2[5]	Mortality & reduced weight	0.07	0.3[5]	2
0.6 (NG)	Selenite[4]	112	11-12	4.0-4.5[6]	Kidney damage & reduced growth	0.6-0.7	0.2[6]	3
Chinook salmon								
4.2 (NG)	SLD[7]	34	26[8]	8.4[8]	Reduced migration	2.0[8]	1.2[8]	4
~1 (swimup)	SLD[7]	90	9.6	6.5	Mortality	1.0	0.8	5
~1 (swimup)	SEM[9]	90	9.6	5.4	Mortality	1.0	0.8	5
~1 (swimup)	SLD[7]	90	5.3	4.0	Reduced growth	1.0	0.8	5
~1 (swimup)	SEM[9]	90	18.2	10.8	Reduced growth	1.0	0.8	5
Fathead minnow								
0.12 (60 day)	Mix[10]	98	15	5.4	Reduced weight	0.4	2.7	6
0.0001 (sw-up)	Rotifer[11]	7-9	55-70	43-61	Reduced weight	NG	NG	7
Striped bass								
251 (NG)	Fish[12]	80	39	15[8,13]	Mortality	1.3[8]	4.4[8]	8
Bluegill								
2.8 (NG)	Mayfly[14]	44	54	31[8,13]	Mortality	2.4[15]	7.6	9
0.2 (3 mo)	SEM[9]	60	6.6	4.2[16]	Mortality	0.7	1.0[16]	10

Table 1. Continued.

Exposure route, species, weight (g), age	Se form	Exp. period (day)	Treated: Se exp. conc. (μg/g; μg/L)	Treated: W-B Se (μg/g)	Effect	Control/Reference: Se exp. conc. (μg/g; μg/L)	Control/Reference: W-B Se (μg/g)	Ref.[1]
Razorback sucker								
~0.005 (5 day)	Zooplt[17]	30	2.4-5.1	3.6-8.7	Mortality	2.3-2.5	3.7-14.3[18]	11
Water								
Rainbow trout								
0.08 (sacfry)	Selenite	60	47	5.2[8]	Mortality & reduced length	<0.4	2.3[8]	12
Chinook salmon								
0.3 (NG)	Mix[19]	60	69	3.8	Mortality	0.9	1.2	4, 13
0.3 (NG)	Mix[20]	60	143	4.9	Reduced growth	0.9	1.2	4, 13
Egg/alvin[21]	Mix[22]	60	67	4.5	Mortality & reduced growth	1.4	2.0	4, 13
Bluegill								
0.3 (5 mo)	Mix[23]	60	640	5.1	Mortality	20	1.0	10
Razorback sucker								
NG (7 day)	Mix[24]	60	480	21	Mortality	<3	1.2	14
NG (7 day)	Mix[24]	60	252	12	Reduced growth	<3	1.2	14
Bonytail								
NG (6 day)	Mix[24]	60	1232	28	Mortality	<3	1.1	14
NG (6 day)	Mix[24]	60	532	17	Reduced growth	<3	1.1	14
Diet and Water								
Bluegill								
NG (2 yr)/	SEM[9]	140 ad/	33.3D/10W	18.7	None	0.8D/0.6W	1.0	15

NG (swimup)	30 lar	33.3D/9W	~6.0	Mortality	0.8+2.7[25]	3.3[26]

[1]References: 1 Goettl & Davies 1978; 2 Hilton et al. 1980; 3 Hilton & Hodson 1983; 4 Hamilton et al. 1986; 5 Hamilton et al. 1990; 6 Ogle & Knight 1989; 7 Bennett et al. 1986; 8 Coughlan & Velte 1989; 9 Finley 1985; 10 Cleveland et al. 1993; 11 Hamilton et al. 1996; 12 Hunn et al. 1987; 13 Hamilton & Wiedmeyer 1990; 14 Hamilton et al. 1998; 15 Coyle et al. 1993.

[2]Selenite incorporated in standard Colorado trout diet.

[3]NG: not given.

[4]Selenite incorporated in a casin-torula yeast trout diet.

[5]Derived from figure 2 in Hilton et al. (1980).

[6]Carcass.

[7]SLD: western mosquitofish (*Gambusia affinis*) collected from San Luis Drain, CA, used as fish meal portion in an Oregon moist pellet diet.

[8]Reported as wet weight and converted to dry weight assuming 75% moisture.

[9]SEM: selenomethionine incorporated into an Oregon moist pellet diet.

[10]Mix: 25% selenomethionine, 25% selenate, and 50% selenite incorporated in a fish food diet.

[11]Rotifer: rotifers fed selenium-laden algae.

[12]Fish: red shiners (*Notropis lutrensis*) weighing about 1 g each were sieved weekly from Belews Lake, NC, where they were chronically exposed to 10 μg/L selenium and food-chain selenium under natural conditions.

[13]Muscle tissue.

[14]Mayfly: burrowing mayfly nymphs (*Hexagenia limbata*) collected from Belews Lake, NC.

[15]Meal worm (*Tenebrio molitor*).

[16]Derived from figure 3 in Cleveland et al. (1993).

[17]Zooplankton: zooplankton collected from Sheppard Bottoms pond at Ouray NWR, UT, water <2 μg/L.

[18]100% mortality.

[19]Mix: 3,023 μg/L boron, 96 μg/L molybdenum, 69 μg/L selenium, and water simulating the San Joaquin River, CA.

[20]Mix: 6,046 μg/L boron, 193 μg/L molybdenum, 143 μg/L selenium, and water simulating the San Joaquin River, CA.

[21]Exposed eyed egg 2 weeks before hatch and alevins 90 days posthatch.

[22]Mix: 2,692 μg/L boron, 92 μg/L molybdenum, 67 μg/L selenium, and well water at Yankton, SD.

[23]Mix: 6:1 mixture of selenate:selenite (measured 550 μg/L selenate, 90 μg/L selenite).

[24]Mix: ratio of environmental concentrations: 2 μg/L As, 630 μg/L B, 10 μg/L Cu, 5 μg/L Mo, 59 μg/L Se (6:1 selenate:selenite), 33 μg/L U, 2 μg/L V, 20 μg/L Zn.

[25]Dry diet contained 0.8 μg/g and brine shrimp nauplii contained 2.7 μg/g; water exposure was 9 μg/L (10:1 selenate:selenite).

[26]75% mortality.

7. As can be observed in Table 1, a wide range of dietary and water borne concentrations have been used n exposures and some exposures have involved mixtures with other trace elements that would have contributed to the observed toxicity. The selenium concentrations in the waterborne exposures are very high relative to the current standard. The main point of the table is that the whole-body residues of selenium, regardless of the exposure route, associated with adverse effects is consistently at 4-5 μg/g.

Likewise, the whole-body concentration in control or reference fish is consistently at 2 μg/g. In the four exceptions shown in the table, one study had control fish that were very old and large (251 g subadults) that were able to accumulate selenium without adverse effects (see reference 8), a second study had relatively large juveniles (2.8 g) that also accumulated selenium without effects (reference 9), a third study very young control fish that had 75% mortality (reference 11), and a fourth study had reference fish that had 100% mortality (reference 12). In each of these studies selenium concentrations were sufficiently elevated to potentially cause adverse effects based on tissue residue concentrations of selenium -- in the studies with older fish, no effects were observed as would be expected, but in the two studies with younger fish, adverse effects were observed.

The difficulty in pursuing a tissue-based criterion is linking water concentrations to food organism (plant, animal, or detritus) to adverse effects on aquatic life. I believe it would be fruitless to try to relate waterborne toxicity studies to a tissue-based criterion because of the great disparity between waterborne and dietary toxicities of selenium. The problem, then is in relating water concentrations to food chain concentrations to whole-animal residues.

Several studies have shown that water concentrations of selenium can be below 2 μg/L, yet food chain concentrations can be elevated to toxic concentrations. Hamilton et al. (1996) reported that water and zooplankton concentrations at four sites at Ouray NWR, UT, over a 4-week period contained were as follows:

Site	Water	Zooplankton
	μ g/L	μg/g
S1	0.7-<1.1	2.3-3.7
S3	0.4-<1.1	4.5-6.7
S4	0.3-<0.6	2.4-5.0
S5	0.6-3.1	12.0-25.7

The selenium concentrations in zooplankton from sites S1, S3, and S4 contain sufficient selenium to be of concern, and based on other biological effect studies, should probably have caused effects. Fish fed these zooplankton had 100 % mortality in less than 2 weeks. The concentrations in zooplankton from S5 are extremely elevated and yet the waterborne concentration was less than the current criterion.

As long as 20 years ago, lakes in Colorado were identified with low waterborne concentrations of selenium from 0.7 to 2.2 μg/L, but high selenium concentrations in aquatic invertebrates of 4.2 to 28.4 μg/g (Birkner 1978). More recently, others have reported situations where waterborne concentrations of selenium were <3 μg/L, but aquatic invertebrates contained potentially toxic concentrations of selenium >3 μg/g (Hallock et al. 1993, Skorupa and Ohlendorf 1991, Zhang and Moore 1996), based on the proposed toxic dietary threshold (Lemly 1993). Consequently, low waterborne concentrations of selenium may be misleading in predicting potential adverse effects on aquatic organisms. A tissue-based criterion would integrate both water, sediment, and dietary exposures, and would be directly relatable to adverse effects on aquatic organisms.

8. There may not be a major difference in uptake rates in sediments for selenium species. Graham et al. (1992) showed that sediments took up radio-labelled selenite and selenomethionine in a similar rapid manner, but during the growing season was lost from the sediment back to the water. Other components of the freshwater experimental ponds where they conducted their study also rapidly taken up by periphyton, pond wed, snails, and *Gambusia*; the most rapid uptake in the selenomethionine pond. They concluded that sediments may at first act as a sink for selenium, but can be remobilized and taken up by biota and the water column (they reported a two-fold increase in organic and/or cation fraction in the water column).

9.

13. Ecosystem type can have a major bearing on the chronic toxicity of selenium to aquatic organisms. However, identifying effects in a lotic (flowing) system may be very difficult compared to measuring effects in a lentic (still) system because of demographically open fish populations (populations that can have recruitment by immigration of individuals from outside an area affected by a toxic stress). Skorupa (1998) has discussed this point, and also emphasized the importance of considering offstream impacts from instream sources of selenium. Identifying adverse effects in lotic environments from environmental degradation is possible if the appropriate approach is used such as (1) indicator taxa or guilds, (2) indices of species richness, diversity, and evenness, (3) multivariate methods, and (4) the index of biotic integrity (IBI); these methods were reviewed by Fausch et al. (1990). They repeated for each of the approaches the necessity of appropriate reference sites and the setting of a priori values, such as expected fish community for a relatively unperturbed stream in a specific ecoregion by a competent fish ecologist/biologist/ichthyologiest. These methods have been intensively used in Ohio (Ohio EPA 1988) and the Midwest, but elsewhere.

Two recent papers (Canton and Van Derveer 1997, Van Derveer and Canton 1997) argued for a site-specific sediment-based criterion for selenium in lotic ecosystems and used as part of their justification the presence of healthy fish populations in the streams they were concerned about. Hamilton and Lemly (1998) noted in their review of the two papers that no methodology was given to substantiate that environmental stresses had not altered the fish community or demonstrate that the

presence fish community was actually healthy. Hamilton and Lemly (1998) also discuss the problem of allowing high instream selenium standards and the high potential for offstream, i.e., backwater, oxbow, or reservoir, effects from bioaccumulation through the food chain.

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Gerhardt Riedel

Gerhardt F. Riedel

-1) Besides selenite and selenate, which other forms of selenium in water are toxicologically important with respect to causing adverse effects on freshwater aquatic organisms under environmentally realistic conditions?

At present, selenate and selenite are the only forms of selenium for which enough combined field analytical data and laboratory toxicity and bioaccumulation work exist to determine that adverse effects are being caused at environmentally realistic conditions. This is not to deny the existence of organic forms of selenium are present in water. A wide variety of organic Se compounds undoubtedly exist in fresh waters, including individual seleno-amino acids such as selenomethionine and selenocysteine, polypeptides of various lengths containing seleno-amino acids, transformation products such as selenonium compounds and dimethyl selenide (DMSe) and dimethyl diselenide (DMDSe) (Cooke and Bruland, 198?;) , and possibly complexes of selenium with dissolved organic matter (DOM). While the seleno-amino acids have been detected in impacted fresh waters, and these compounds are often observed to be highly bioaccumulative and toxic (Kiffney and Knight, 1990; Riedel et al., 1991), these compounds are present in vanishingly small concentrations, are difficult to determine, and are extremely labile. These compounds are also formed biologically in response to enrichment with selenate and selenite (Besser, et al., 1994), and are thus closely linked to the concentrations of the inorganic species, thus regulating their concentrations should also regulate the formation of the amino-acids as well. Other known dissolved species such as, selenonium compounds, and (DMSe) and (DMDSe), are likely to be less toxic than the inorganic species, although to my knowledge, this has not been tested in aquatic systems.

Confidence level: High

I-2) Which form (or combination of forms) of Se in water are most closely correlated with chronic effects)

Since both selenate and selenite can be present essentially separately, or combined in mixtures depending on the source of the selenium and subsequent transformations, and because the two forms have substantially different patterns of bioaccumulation and toxicity (e.g.Kiffney and Knight, 1990; Riedel et al., 1991, Maier and Knight, 1993) , it is imperative that analytical methods used to evaluate Se in aquatic systems be capable of separating selenate, selenite (Cutter 1978). In most cases, the concentration of Se in particles is small with respect to the concentration of Se in the dissolved phase, and the difference between total dissolved and total recoverable Se is unimportant, and may be difficult to resolve analytically. Furthermore, it is of greater interest whether Se in the suspended particles is elevated compared to total particle mass or organic carbon (Cutter, 1985). However, it also is important to distinguish between the inorganic forms which are likely the primary forms for uptake (at the lower trophic levels) and toxicity, and the myriad organic forms which have been produced in response to inorganic Se enrichment, and probably represent a net reduction in toxicity (e.g. Yu et al., 1997). This separation can also be done within the analytical framework of Cutter (1978). At this time it is unlikely that the organic Se species will be better resolved analytically at a regulatory level, although this should be an area of continued research.

Confidence level: High

I-3A) In priority order, which water quality characteristics (e.g. pH, TOC, sulfate, interactions with other metals such as Hg) are most important in affecting the chronic toxicity and bioaccumulation of Se to freshwater aquatic life under environmentally realistic conditions?

The answers differ for selenate and selenite. For selenate, sulfate appears to act as a competitive inhibitor of both uptake and toxicity in a variety of plants and animals (Wheeler et al., 1982, Riedel et al and Sanders, 1996; Ogle and Knight, 1996) . Given that areas where selenate is mobilized (e.g.

agricultural drain water, and various types of geology) are also generally sites where sulfate is enriched as well, sulfate should be considered important in the environmental toxicity of selenate. Arsenic (As) and molybdenum (Mo), which are also mobilized under similar conditions, appear to produce additive toxicity with selenate, and should also be considered important (e.g. Naddy et al., 1995). TOC, DOC, pH and other common water quality characteristics do not appear to significantly influence the environmental toxicity of selenate.

It appears likely, based on simple chemical principals, that pH has a significant effect on the speciation of selenite over the environmentally reasonable pH range, and thus is likely to exert strong influences on the uptake and toxicity of selenite in a variety of organisms (Riedel and Sanders, 1996). Unfortunately, fairly little work has been done on this relationship, and some additional work would be necessary to show its generality across species. For algae, there is some evidence that selenite bioaccumulation can be affected by phosphate concentrations (Riedel and Sanders, 1996). This is both an environmental concern, as the phosphate status of a freshwater system can be quite variable, as well as in evaluating reports of selenite accumulation and toxicity to algae in the laboratory, which is commonly done at environmentally unrealistic concentrations of phosphate. Again, as with pH, the evidence for this effect is limited, and it should be further verified. It is likely that other similar elements (arsenate, molybdate etc) and possibly mercury can influence selenite uptake and toxicity under environmentally realistic conditions although this work has focused on selenate dominated systems. Other water quality parameters, such as TOC, hardness, sulfate, have not been shown to substantially influence selenite accumulation and toxicity.

Confidence level: medium

I-3B) Of these which have been or can be quantitatively related to selenium chronic toxicity or bioaccumulation in aquatic organisms. How strong or robust are these relationships?

The relationship between sulfate and selenate is at least semi-quantitative and relatively robust (particularly at the algal level). However, at higher trophic levels, this relationship is likely to be not a strong, and to be expressed indirectly through the food web.

Confidence level: medium

I-3C) How certain are applications of toxicity relationships derived from acute toxicity and water quality characteristics to chronic toxicity situations in the field?

Relatively poor. Although algae appear to have relatively uniform responses to concentrations within a species or group, there are always algae that grow under extreme conditions and thus it is difficult to determine that high selenium concentrations have any toxic effect on algae in the field. Toxicity to algae is more likely to show as changes in the species composition of algal communities rather than changes in growth or biomass. The influence of sulfate on the uptake and toxicity of selenate in Crustacea (cladocera and midge larvae) is somewhat variable, and there appears to be no systematic effect with fish.

Confidence level: medium

II. - 4) Which forms or selenium in tissues are toxicologically important with respect to causing adverse effects of freshwater organisms under environmentally realistic conditions and why?

It is widely held that the accumulation of selenium in selenoamino acids and selenoproteins is the predominant form of Se in tissues and that they are responsible for most of the effects of selenium observed in organisms, either good or ill. Although I do not actively collect that literature, I know of no

evidence of different forms of selenium within tissues that act in different manners. However, it is a reasonable presumption that selenate and selenite that had recently entered a tissue and had not yet been converted to an organic form might have different effects. Similarly, degradation products such as selenonium compound and DMSe and DMDSe are likely to have substantially different toxicological properties than their parent organic compounds; but tissue measurements of these compounds are virtually unavailable. Unfortunately, speciation analysis of selenium in a tissue matrix is very difficult.

Confidence level: low

II - 5) Which form (or combination of forms) of selenium in tissues are most closely correlated with chronic effects on aquatic life in the field?

Following the answer above, I am unaware of any correlations of Se tissue speciation with chronic effects of Se. However, reasoning from chemical principals, I would prefer analytical techniques that could 1) separate inorganic Se (particularly selenite) from "incorporated" Se; 2) determine Se incorporated into proteins, and preferably which seleno-amino acids were incorporated into which proteins, in which tissues, and 3) separate and identify the "breakdown" products, selenonium compounds, DMSe and DMDSe.

Confidence level: low

II - 6) Which tissue (and in which species of aquatic organisms) are best correlated with overall chronic toxicological effect thresholds of selenium?

Liver and gonad tissue are relatively responsive to Se concentrations in fish, while muscle tissue responds relatively slowly. Although I am not aware of any such research one would suspect that the hepatopancreas of larger crustaceans would also be relatively responsive. Observed chronic Se toxicity

in natural aquatic systems has been largely restricted to fish, and among fish, largely restricted to centrarchids, although some suckers and salmonids may be affected as well.

Confidence level: medium

II - 7) How certain are we in relating water-column concentrations of selenium to tissue-residue concentrations in top trophic-level organisms such as fish? What are the primary sources of uncertainty in this extrapolation?

We are fairly uncertain in predicting Se in fish tissues from water column Se concentrations. Given that we can make a rough projection of phytoplankton and zooplankton concentrations from form specific water column data, and other water quality data, (and that projection is likely to be quite rough, see attached Fig. 1), the transfer of that Se to fish can vary with the type of fish, the feeding behavior of the fish at any particular time. When you also consider a variable benthic food chain for most of the important fish (centrarchids, salmonids and suckers) I believe it will become quite difficult to predict fish tissue concentrations without a significant modeling effort.

Confidence level: medium

III - 8) Which forms of selenium in sediment are toxicologically important with respect to causing adverse effects on freshwater aquatic organisms under environmentally realistic conditions?

In aerobic sediment, most of the Se is likely to be present as either adsorbed

selenite or as organic Se present in the microbial compartment. Se in the organic form is likely have a higher assimilation efficiency, and thus be more available for toxicity than inorganic Se adsorbed to particles. Furthermore, in aerobic sediments, pore water concentrations of Se may be high enough that direct uptake of selenite and selenate by benthic organisms could be a significant route. In anaerobic

sediment, elemental Se (Se^o) is likely to be the predominant form, and pore water Se is likely to be vanishingly small. While there is evidence that Se^o is available to molluscs (Luoma et al., 1992), it seems likely that it is less available than organic Se, and it is also likely to be much less available to benthic animals with short digestion times, such as chironomids, and oligochaetes.

Confidence level: high

III - 9) Which form or forms in sediment are most closely correlated with chronic effects on aquatic life in the field?

Following the discussion above, I would like to see analytical techniques for Se in sediment to separate organically bound Se, adsorbed selenate and selenite, and Se^o.

Confidence level: high

III - 10) In priority order, which sediment quality characteristics (e.g. TOC) etc. are most important in affecting the chronic toxicity and bioaccumulation of selenium to freshwater aquatic life und environmentally realistic conditions? Of these, which have been (or can be) quantitatively related to selenium chronic toxicity or bioaccumulation in aquatic organisms?

In priority order, oxidation state (Eh, presence of sulfide, etc), TOC, pH, grain size. Indirectly, the redox condition can be linked to the concentrations of Se in fish in Hyco reservoir, which in turn links to the chronic toxicity of Se. TOC in sediment has been quantititatively linked to accumulation of Se in fish in riverine systems.

Confidence level: medium

III - 11) How certain are we in relating water-column concentrations of selenium to sediment concentrations? What are the primary sources of uncertainty in this extrapolation?

Very uncertain. Water column concentrations of Se can fluctuate over a wide range in a relatively short period of time, where sediment accumulation integrates Se inputs over a very long period. Furthermore, the processes that transfer Se from the water column to the sediment and vice versa can operate at very different directions and rates depending on (roughly in order of importance) selenium form, redox conditions, productivity, benthic fauna, temperature.

Confidence level: High

IV - 12) How does time variability in ambient concentrations affect the bioaccumulation of selenium in aquatic food webs, and in particular, how rapidly do residues in fish respond to increases and decreases in water concentrations?

The effect time variability of ambient concentrations of Se on bioaccumulation can be expected to depend on the time constants of individual steps. For a water column-based food chain, phytoplankton and zooplankton would respond relatively quickly to changes in water column Se concentrations, and fish would respond to changes in prey concentrations according to their own assimilation and depuration kinetics (on the order of months). Large piscivores would presumably have a longer response time due to the lag in their prey. Once a substantial component of benthic food is assumed, the link between ambient water concentrations and fish accumulation is seriously compromised, since high concentrations of Se in the sediment can lead to substantial accumulation in fish despite relatively low water column concentrations of Se.

Confidence level: medium

IV -13) To what extent would the type of ecosystem (e.g. lentic, lotic) affect the chronic toxicity of selenium?

The type of ecosystem could have a dramatic effect on the chronic effect of Se loadings. In a relatively quickly moving body of water, such as a stream, river, or short residence time lake, the rapid overturn of water and sediment could prevent the accumulation of substantial quantities of Se in the sediment. In a long-term residence time lake, particularly one with a seasonally anoxic hypolimnion, Se could be extensively trapped by the sediment, which could be a continuing source for chronic toxicity through the benthic food web, and seasonal releases of Se to the water column.

Confidence level: medium

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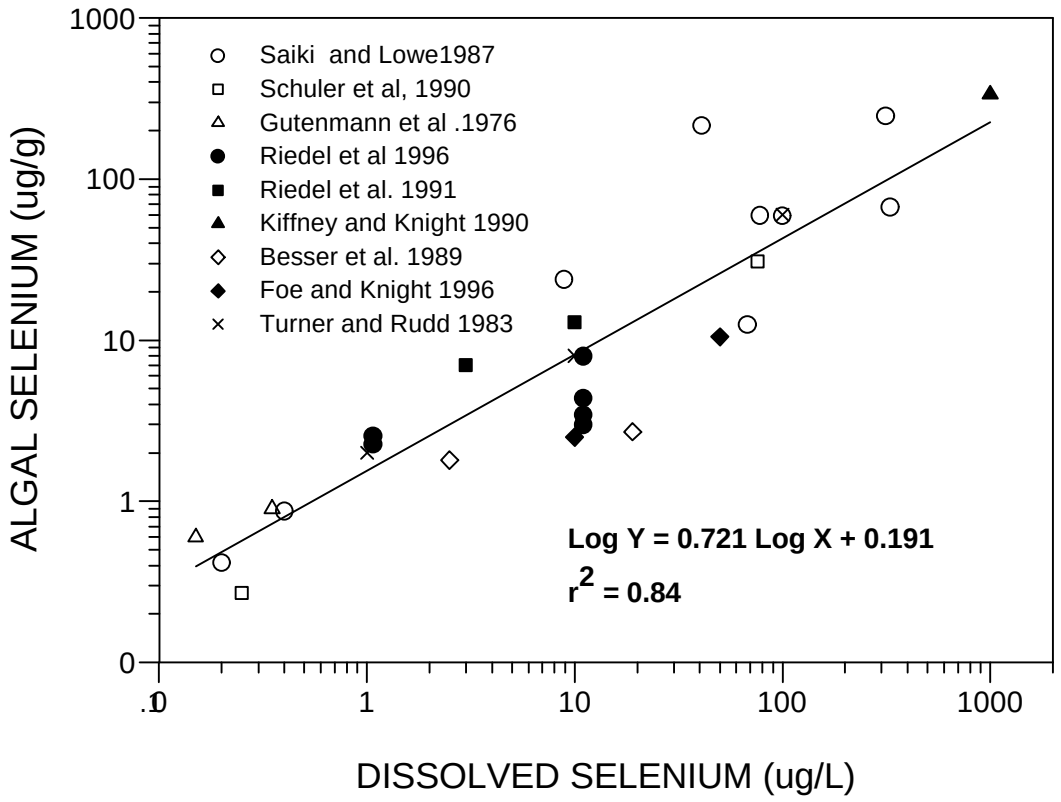

Figure 1. Concentrations of Se in algae compared to the concentrations of total Se in the water for a number of freshwater ecosystems and laboratory experiments.

Joseph Skorupa

Peer Consultation Workshop on Selenium Aquatic Toxicity

and Bioaccumulation

Premeeting Written Comments

1. *Besides selenite and selenate, which other forms of selenium in water are toxicologically important with respect to causing adverse effects on freshwater aquatic organisms under environmentally realistic conditions?*

Another form of selenium in water that appears to be toxicologically important is selenomethionine (and perhaps other selenoamino acids). Se-meth has repeatedly been demonstrated to exhibit order-of-magnitude higher bioaccumulation dynamics (e.g., Riedel et al. 1991; Graham et al. 1992; Besser et al. 1993; Rosetta and Knight 1995) and toxicity (e.g., Ingersoll et al. 1990; Kiffney and Knight 1990; Maier and Knight 1993; Maier et al. 1993) than selenite or selenate. Maier and Knight (1993) caution, however, that species-specific exceptions to the general trend also occur.

Ingersoll et al. (1990) reported a chronic geometric mean-maximum acceptable toxicant concentration (GM-MATC)for Se-meth of 0.16 ug Se/L for *Daphnia magna*. Furthermore, based on observations that fish foods containing 3-20 mg Se/kg, on a dry weight basis, are sufficiently contaminated to cause toxic effects (depending on the life stage and species of fish being considered) (e.g., Hamilton et al. 1990; Lemly 1993a, 1997a), and based on a bioconcentration factor of 382,000 for Se-meth from water to *zooplankton* (at 0.1 ug Se/L; Besser et al. 1993), as little as 0.008-0.052 ug Se/L in the form of Se-meth appears sufficient to produce a toxic diet for fish. Because bioconcentration factors are inversely proportional to waterborne concentrations (e.g., Besser et al. 1993), the bioconcentration factor for Se-meth at 0.008-0.052 ug Se/L may actually be greater than the 382,000 observed at 0.1 ug Se/L and therefore waterborne concentrations of less than 0.008-0.052 ug Se/L may actually be sufficient to result in toxic diets for fish.

Given such evidence that Se-meth concentrations in water on the order of parts per trillion are sufficient to be toxicologically significant, it is important to assess how commonly, and under what conditions, such concentrations would be met or exceeded in the field? That's a question I would need more time to research the literature on. I suspect that other panel members are probably already familiar with that segment of the selenium literature and can address that issue.

2. *Which form (or combination of forms) of selenium in water are most closely correlated with chronic effects on aquatic life in the field? (In other words, given current or emerging analytical techniques, which forms of selenium in water would you measure for correlating exposure with adverse effects in the field?) Note: Your response should include consideration of operationally defined measurements of selenium (e.g., dissolved and total recoverable selenium), in addition to individual selenium species.*

I am not aware of sufficient research on this topic, with regard to aquatic life, to provide a definitive, or even putative, answer.

Conceptually, waterborne selenium concentrations are useful for predicting chronic effects only to the extent that they consistently covary with the partitioning of selenium into the aquatic food chain. Although consistent covariation of waterborne selenium and foodchain selenium has been reported on a local scale for physically uniform and biotically simple aquatic systems (e.g., Birkner 1978; Skorupa and Ohlendorf 1991), at a national scale any form of waterborne selenium (or combination of forms) likely would be relatively unreliable for predicting chronic effects. This is because at a national scale the immense variety of environmental permutations and site histories would commonly include cases where waterborne selenium concentrations and food chain selenium concentrations are discordant (e.g., Luoma et al., 1992; Lemly 1997b; Setmire and Schroeder 1998; Maier et al., In Press). For example, waterborne concentrations of selenium can be low either because of low mass loading into a water body (in which circumstance foodchain selenium will also be low and nonhazardous) or because of efficient and rapid partitioning of elevated mass loads out of the water column and into other compartments, including the food chain (a potentially hazardous situation). Thus, *a priori,* a low waterborne concentration of selenium isn't necessarily safe or hazardous.

Although measures of total selenium on both a filtered (dissolved) and unfiltered (total recoverable) basis will be uncertain predictors of chronic effects at a national scale, based on first principles it can be deduced logically that unfiltered values (total recoverable) should usually be better predictors than filtered (dissolved) values. It is selenium contamination of the aquatic food chain that most directly causes chronic effects in sensitive aquatic taxa (such as fish; e.g., Hermanutz et al. 1992), and since a substantive part of the particulate load in a water column is often composed of foodchain tissues (living and decaying), it logically follows that including particulates in measures of waterborne selenium should usually increase the linkage between such measures and the probability of chronic effects.

3. ***A) In priority order, which water quality characteristics (e.g., pH, TOC, sulfate, interactions with other metals such as mercury) are most important in affecting the chronic toxicity and bioaccumulation of selenium to freshwater aquatic life under environmentally realistic exposure conditions?***

B) Of these, which have been (or can be) quantitatively related to selenium chronic toxicity or bioaccumulation in aquatic organisms? How strong and robust are these relationships?

C) How certain are applications of toxicity relationships derived from acute toxicity and water quality characteristics to chronic toxicity situations in the field?

A) I am unaware of any studies that have simultaneously partitioned the relative proportions of variance in selenium bioaccumulation explained by pH, TOC, sulfate concentration, and chemical interactions effects. Birkner (1978) simultaneously examined several water quality parameters such as dissolved sulfate, hardness, and conductivity and found that none of those parameters explained a significant amount of variation in foodchain bioaccumulation of selenium across thirty field sites surveyed in Colorado and Wyoming. Foodchain selenium did, however, significantly covary with water and sediment concentrations of selenium. I suspect that there is insufficient basis in the scientific literature to conclusively establish a priority order for water quality characteristics that

influence selenium toxicity.

There is increasing evidence, however, that interaction effects with mercury may rank prominently among the factors influencing selenium toxicity. For example, Heinz and Hoffman (1998) recently demonstrated that the most environmentally relevant forms of dietary selenium and mercury (selenomethionine and methyl-mercury) caused highly synergistic reproductive toxicity to an egg-laying vertebrate. Similarly, studies at a set of mercury-contaminated lakes in Sweden provided circumstantial field evidence for severe toxic effects on fish populations at unexpectedly low levels of experimental selenium additions (3-5 ug Se/L as sodium selenite; Paulsson and Lundbergh 1991).

B) Concentrations of dissolved sulfate have been demonstrated in short-term bench top experiments to strongly inhibit bioconcentration and bioaccumulation of dissolved selenate by algae and aquatic invertebrates (e.g., Hansen et al. 1993; Maier et al. 1993; Williams et al. 1994; Ogle and Knight 1996). These studies have demonstrated that such inhibition can be quantitatively related to selenium bioaccumulation and toxicity, at least within the context of biotically and chemically simplistic experimental conditions.

C) The bench top studies of sulfate interaction effects cited above, however, do not appear to translate very well to observed patterns of selenium bioaccumulation (and therefore chronic effects) in the field. As already mentioned, Birkner (1978) found no significant effect of dissolved sulfate concentrations (5 to 9,611 mg/L) on foodchain bioaccumulation in the field. Likewise, very strong correlations between waterborne selenium and aquatic foodchain selenium (correlation coefficients > 0.9 for most foodchain taxa) were observed across multiple field sites in California's Tulare Lake Basin despite widely varying concentrations of dissolved sulfate ranging from 2,000 to 100,000 mg/L (Skorupa and Ohlendorf 1991; Skorupa 1998). This pronounced discrepancy between lab and field observations may be related to the fact that although selenate and sulfate compete for a common uptake pathway, bioaccumulation of selenite and selenomethionine occurs via separate pathways

from sulfate (e.g., Maier et al. 1993).

Even in the presence of high sulfate concentrations, uptake of selenate and its biotransformation to reduced forms of selenium such as selenite and selenomethionine by algae and other primary producers is only depressed, not eliminated. Also, selenate-reducing bacteria in sediments and water can function independently of sulfate concentrations (e.g., Oremland et al., 1989; Gerhardt et al., 1991). Bacteria play a critical role in the foodchain cycling of selenium (e.g., Bowie et al., 1996). Thus, over time, even if the original form of selenium entering an aquatic system was virtually pure selenate, reduced forms of selenium that are not inhibited by sulfate (e.g., selenite, selenomethionine, etc.) increasingly become available and increasingly dominate the process of foodchain bioaccumulation.

For example, drainage water in the San Joaquin Valley of California was found to contain selenium as selenate, selenite, and selenomethionine (Se-Meth) in a ratio of approximately 18:3:1 (Besser et al. 1989). Bioconcentration factors for periphyton, however, showed a reverse ratio of about 1:6:120 (Besser et al. 1989). Thus, the approximate ratio of selenium uptake from selenate, selenite, and Se-Meth would be 18:18:120. Therefore, only about 11% (18/156) of bioaccumulated selenium in the periphyton would be taken up directly from the inventory of dissolved selenate. Under these circumstances, even if a sulfate-interference effect as high as 50% assimilative inhibition were occurring it would cause only about a 5% (0.5 x 0.11) reduction in overall bioaccumulation of selenium. At toxic threshold exposures in the region of 2-5 ug/L waterborne selenium, a 5% reduction would be very negligible in absolute terms. Even this example probably overestimates the contribution of selenate to overall bioaccumulation of selenium because it does not account for the cumulative loading of predominantly non-selenate species of selenium into aquatic sediments, which is another major bioaccumulation pathway that further devalues the relative importance of dissolved selenate selenium. It is quite plausible that in biologically and chemically complex aquatic environments, even where concentrations of dissolved sulfate are low, only a minute proportion of selenium bioaccumulation is due to direct uptake of selenate selenium. Recently, Milne (1998) has

similarly noted that because of the vast differences in assimilatory dynamics of selenate versus reduced forms of selenium (unrelated to uptake inhibition by sulfate)"...*selenate is often a spectator in living systems*."

It appears that 48-hr to 96-hr bench top experiments may be too short in duration and too biotically simplified to mimic the typical real-world progression from a selenate-dominated water to a complex mixture of multiple chemical species of selenium metabolically dominated by reduced forms of selenium whose cycling is independent of dissolved sulfate concentrations. In summary, for whatever reasons, field data for aquatic organisms do not support the notion that foodchain bioaccumulation of selenium is sulfate-dependent. Sixty years ago, Beath (1937) concluded that the "...*sulfur-selenium antagonism theory has not been found generally applicable to farm and range practices* [for ameliorating selenium toxicity to range animals] *of the Rocky Mountain region*."

4. *Which forms of selenium in tissues are toxicologically important with respect to causing adverse effects on freshwater aquatic organisms under environmentally realistic conditions and why?*

Several studies have concluded that the most common forms of dissolved selenium all appear to be metabolically transformed to a common pool of selenium (a uniform toxicological currency) in foodchain biota (e.g., Gissel-Nielsen 1987; Zayed and Terry 1992; Besser et al. 1993; Milne 1998). Several studies have also indicated that the putative universal foodchain currency is either selenomethionine or one or more forms of selenium with toxicological profiles functionally equivalent to selenomethionine (e.g., Woock et al. 1984; Hamilton et al. 1990; Heinz 1996). Thus, it appears that one or more selenoamino acids, possibly including selenomethionine, are the toxicologically important forms of selenium in biotic tissues.

It has been suggested that selenoamino acids are toxicologically important because of their propensity to substitute for the analogous sulfuramino acids and thereby alter the normal structure and function of proteins (e.g., Rosetta and Knight 1995; Lemly 1997a; Martens and Suarez 1998).

5. *Which form (or combination of forms) of selenium in tissues are most closely correlated with chronic effects on aquatic life in the field? (In other words, given current or emerging analytical techniques, which forms of selenium in tissues would you measure for correlating exposure with adverse effects in the field?)*

This question is still largely unaddressed in the scientific literature. Much of my response provided above for question number 4 is also relevant here. Therefore, it is likely that prediction of chronic effects in the field could be improved by examining the relationship of specific selenoamino acids to chronic effects. Nonetheless, even on the basis of undifferentiated total selenium concentrations in tissues, exposure-response relationships strong enough to provide good predictive value for chronic effects in the field have been delineated (e.g., Lemly 1993b, 1997a). In some respects, the crucial question is not what specific form of selenium to measure in tissues, but rather what's the most appropriate tissue to focus on for measures of total selenium or specific forms of selenium.

Reproductive tissues, such as fish ovaries or eggs, have clearly been established as the answer to the latter question (e.g., Lemly 1993c, 1995, 1996a), as has been established for aquatic birds also(Heinz 1996; Skorupa 1998).

6. *Which tissues (and in which species of aquatic organisms) are best correlated with overall toxicological effect thresholds for selenium?*

Reproductive tissues of sensitive species of fish, such as centrarchids and salmonids (e.g., Lemly 1996b, 1997b).

7. *How certain are we in relating water-column concentrations of selenium to tissue-residue concentrations in top trophic-level organisms such as fish? What are the primary sources of uncertainty in this extrapolation?*

It is much easier to reliably relate foodchain concentrations of selenium to fish tissue residues than to reliably relate water-column concentrations because water-column selenium occurs in several toxicological currencies, whereas foodchain selenium occurs in a reasonably universal toxicological currency (see response provided above for question number 4). It should be possible with reasonable certainty to experimentally determine levels of foodchain selenium that result in toxicological threshold levels of tissue selenium in fish (e.g., Woock et al., 1987; Hamilton et al., 1990; Cleveland et al., 1993; Coyle et al., 1993). Then for various forms and mixtures of water-column selenium the partitioning dynamics into aquatic foodchain compartments would have to be determined. Ultimately this would identify waterborne concentrations that have the potential to result in toxicological threshold levels of foodchain selenium. When and where such experimentally determined potentials for chronic effects would be realized in the field would still depend on a complex interactive combination of site-specific environmental conditions.

Alternatively, field data systematically collected on a national scale, or at least on a regional scale,

could be analyzed to characterize the realized probabilities of exceeding threshold tissue concentrations in fish that are associated with a range of water-column selenium concentrations. Existing databases such as the National Water Quality Assessment Program (NAQWA), or National Irrigation Water Quality Program (NIWQP) might provide the basis for a preliminary investigation of this nature. This has already been done for eggs of aquatic birds (Adams et al., In Press) associated with irrigation projects in the western United States, revealing that only when waterborne concentrations of dissolved selenium (primarily as selenate) are near or below 1 ug/L is the probability of toxic threshold exceedance below 10%.

Until more experimental data are generated for fish, or probability analyses are conducted on geographically extensive field-collected databases for fish, the relationship between waterborne selenium concentrations and selenium concentrations in fish tissues should be viewed as poorly characterized and therefore relatively uncertain.

Questions 8-12.

I defer on these questions to other committee members. In my view, most issues associated with developing a sediment-based chronic criterion are largely unaddressed in existing scientific literature. As far as I am aware there has yet to be published even a single set of field data reporting selenium concentrations in carefully matched samples of sediment and benthic foodchain fauna, which seems to me like a logical first step toward developing sediment criteria. In any case, I look forward to other committee members' comments on sediment issues.

13. ***To what extent would the type of ecosystem (e.g., lentic, lotic) affect the chronic toxicity of selenium?***

The type of ecosystem would affect the chronic toxicity of selenium to the extent that categorical differences in ecosystems either alter the dynamics of selenium bioaccumulation in the aquatic foodchain, alter the form of selenium incorporated into foodchain tissues, or alter the relative exposure of upper trophic level organisms (i.e., fish) to different foodchain compartments (e.g., benthic versus water-column biota, fauna versus flora, etc.). There are several sound conceptual bases for expecting lotic ecosystems to be less prone to chronic toxicity than lentic ecosystems (Skorupa 1998), however, unless lotic and lentic ecosystems are sufficiently isolated from one another hydrologically, criteria protective of the more sensitive of the two systems would have to be applied to both ecosystems.

For example, in a normal water year, the entire flow of the Colorado River is diverted offstream, thus, criteria applied to the Colorado River (lotic) ecosystem would also have to be protective of offstream (lentic) ecosystems such as the Salton Sea (Skorupa 1998). As another example, many rivers now have large onstream reservoirs that create direct hydrologic linkages of lotic and lentic ecosystems, thus, any criteria implemented for the river would also have to be protective of onstream reservoirs. Because of extensive anthropogenically created linkages of lotic and lentic ecosystems, ecosystem affects on chronic toxicity may be largely a moot technical issue except for identifying baseline criteria that are appropriately protective of the most sensitive ecosystem link.

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William Van Derveer

EER CONSULTATION WORKSHOP ON SELENIUM AQUATIC TOXICITY AND BIOACCUMULATION: PREMEETING COMMENTS

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INTRODUCTION

In preparation for the Peer Consultation Workshop, the U.S. Environmental Protection Agency (EPA) requested that the workshop participants comment on a series of issues related to the aquatic toxicity and bioaccumulation of selenium (Se). The EPA issues and my comments (in italics) are provided below. Regardless of how the revised chronic water quality criteria are ultimately expressed: water-column, tissue, or sediment; I hope that it is based upon sound science and is amenable to site-specific modification where appropriate.

ISSUES AND COMMENTS

I. Technical Issues Associated with a Water-Column-Based Chronic Criterion

1. Besides selenite and selenate, which other forms of selenium in water are toxicologically important with respect to causing adverse effects on freshwater aquatic organisms under environmentally realistic conditions?

Comments: Organic Se compounds such as selenomethionine have been shown to be toxicologically important is some laboratory experiments (e.g., Maier and Knight 1993); however, I am unaware of any studies that have shown these specific organic Se forms to occur in the water column of the natural environment. Some studies (e.g., Cutter 1991; Zhang and Moore 1996) suggest that naturally occurring organic Se forms can be found in the water column; however, the chemical identity and potential toxicity of these compounds are largely unknown.

2. Which form (or combination of forms) of selenium in are most closely correlated with chronic effects on aquatic life in the field?) (In other words, given current or emerging analytical techniques, which forms of selenium in water would you measure for correlating exposure with adverse effects in the field?) Note: Your response should include consideration of operationally defined measurements of selenium (e.g., dissolved and total recoverable selenium), in addition to individual selenium species.

Comments: Waterborne Se concentration alone, regardless of its method of measurement, appears to correlate poorly with chronic effects on aquatic life in the field. Chronic toxicity under field conditions does not result from direct waterborne Se exposure, rather, it results from the propensity for Se to cycle through the food web, the dominant exposure route, and cause reproductive impairment in fish and wildlife. A review of the literature by Lemly (1993a) found that “the consensus of research studies is that most of the Se in fish tissues results from Se in the diet rather than in the water”. Limitations in the ability to extrapolate Se concentrations from water to tissue are discussed in my comments for question 7.

If new water-column-based chronic criteria are ultimately developed, they should account for

selenite and selenate individually as their relative toxicity and bioaccumulative potential differ. Bioaccumulation and subsequent reproductive effects on warmwater fish populations observed at Belews Lake, NC were the result of selenite discharge from fly ash. The current chronic criterion for Se is based on studies conducted at that site and thus is based only on selenite. Studies of outdoor experimental streams in central Minnesota (Hermanutz 1992; Hermanutz et al. 1992; Schultz and Hermanutz 1990) have been cited as validation for the current chronic criterion. In these studies, effects on fathead minnow (Pimephales promelas) and bluegill (Lepomis macrochirus) reproduction and adult survival were observed using waterborne selenite concentrations of 10 and 30 μg/L. Criteria based upon and validated by the Belews Lake and experimental streams studies fail to acknowledge the fact that most waterborne Se in the western U.S. occurs as selenate. In this region, Se entering aquatic ecosystems is primarily derived from weathering of selenate from geologic materials (i.e., Cretaceous marine shale) not industrial discharges.

With respect to operationally defined measurements of waterborne Se, there seem to be only small differences between total and dissolved analyses for most field samples. Seiler (1996) summarized U.S. Department of Interior data from 26 investigations that were conducted throughout the western U.S. and found that total and dissolved Se concentrations were approximately equal at concentrations in excess of 10 μg/L and that moderate variability exists at concentrations below 10 μg/L. Seiler (1996) concluded that the likely source of the observed variability was Se-bearing suspended particulates and cautioned that the difference between total and dissolved Se concentrations may be greatest in highly productive waters where large algal populations are present.

3-A) In priority order, which water quality characteristics (e.g., pH, TOC, sulfate, interactions with other metals such as mercury) are most important in affecting the chronic toxicity and

bioaccumulation of selenium to freshwater aquatic life under environmentally realistic exposure conditions?

Comments: The only water quality characteristic, of which I am aware, that may modify chronic Se toxicity is sulfate. There is some evidence to suggest that the chemical similarities of selenate and sulfate allow sulfate to reduce the acute toxicity of selenate in laboratory exposures. High dissolved sulfate concentrations can substantially reduce the acute toxicity of selenate to aquatic invertebrates (Ingersoll et al. 1990; Maier et al. 1993; Ogle and Knight 1996). Since sulfate appears to reduce the short-term bioavailability of selenate it is plausible that a similar effect may occur during long-term field exposures.

3-B) Of these, which have been (or can be) quantitatively related to selenium chronic toxicity or bioaccumulation in aquatic organisms? How strong and robust are these relationships?

Comments: I am not aware of any studies that have specifically focused on the potential interaction of sulfate and selenate from the standpoint of chronic toxicity or bioaccumulation during long-term exposures.

3-C) How certain are applications of toxicity relationships derived from acute toxicity and water quality characteristics to chronic toxicity situations in the field?

Comments: Extrapolation from acute toxicity data to chronic toxicity situations in the field is inappropriate for Se. Acute Se toxicity is caused by direct waterborne exposure whereas

chronic Se toxicity under field conditions is caused by excessive dietary (food web) exposure. These two exposure pathways are sufficiently distinct as to prohibit valid extrapolation between acute and chronic toxicity data. Moreover, the degree of dietary contamination at a given site is a function of many site-specific factors, which affect the fate and bioavailability of waterborne Se, thus application of a single chronic criterion to all sites may be inappropriate.

II. Technical Issues Associated with a Tissue-Based Chronic Criterion

4. Which forms of selenium in tissues are toxicologically important with respect to causing adverse effects on freshwater aquatic organisms under environmentally realistic conditions and why?

Comments: Selenium in the tissues of aquatic organisms is customarily reported as total Se and is generally considered to be "organic Se" rather than a specific chemical form such as selenomethionine or selenocysteine. The lack of published information regarding the chemical forms of Se in tissues indicates that significant research would be needed to develop a full understanding of this issue.

5. Which form (or combination of forms) of selenium in tissues are most closely correlated with chronic effects on aquatic life in the field? (In other words, given current or emerging analytical techniques, which forms of selenium in tissues would you measure for correlating exposure with adverse effects in the field?

Comments: As noted above, Se in the tissues of aquatic organisms is commonly reported as total Se. Use of total Se data may be appropriate for criteria-setting provided the tissue concentrations that are used to derive the criteria are also based on field exposures. However, it is plausible that the composition of Se forms in the tissues of field exposed organisms may vary depending upon the form of Se that was initially released to the environment (i.e., selenate, selenite, or organic Se compounds). Thus, aggregating data without respect to the released Se form may increase the level of uncertainty associated with tissue-based criteria. Moreover, tissue concentration data developed from chronic laboratory or experimental stream exposures (e.g., Besser et al. 1993; Coyle et al. 1993; Dodds et al. 1996; Hermanutz et al. 1992) may not represent the true composition of Se forms that occur in organisms that are exposed to Se the natural environment. Thus, data derived from these types of studies should not be used for criteria development unless there is a reasonable demonstration that the Se forms that accumulated in tissues of the test organisms were comparable to those found in the natural environment.

6. Which tissues (and in which species of aquatic organisms) are best correlated with overall chronic toxicological effect thresholds for selenium?

Comments: The literature pertaining to this issue is varied, suggesting that under certain conditions, the Se concentrations in an organism's diet, muscle or whole-body, hepatic tissues, and eggs all can serve as reliable predictors of chronic effects on aquatic life. Other workshop participants are better qualified to provide an in-depth response to the issue of specifically which tissues should be measured.

Although it is common practice, the use of whole-body samples to assess Se accumulation is problematic because it assumes that all of the Se contained within an organism has been assimilated. This is not necessarily true because an organism's gut contents can be a mixture of bioavailable and non-bioavailable materials. The true magnitude of Se bioaccumulation can be overstated by assuming that these non-bioavailable materials, some of which may contain Se, have been assimilated by the organism. This issue can be alleviated by clearing an organism's gut through depuration or mechanical means prior to analysis.

In terms of which species of organisms best reflect toxic effects, fish generally appear to be more sensitive to Se exposure than invertebrates. The fish species that are most susceptible to Se toxicity belong to the families Centrarchidae, Clupeidae, Percichthyidae, Percidae, and Catostomidae as indicated by their elimination from Belews Lake, NC shortly after the addition of Se-enriched effluent, whereas, species belonging to the family Cyprinidae were largely unaffected (Lemly 1985a, 1985b, 1993b). It is important to note that gamefish do not occur in all waters and that tissue-residue thresholds derived for gamefish may not be

applicable to more tolerant nongame species. Water quality criteria should account for the relative sensitivity of the biota that are to be protected.

7. How certain are we in relating water-column concentrations of selenium to tissue-residue concentrations in top trophic-level organisms such as fish? What are the primary sources of uncertainty in this extrapolation?

Comments: The relationship between waterborne concentration and tissue-residue concentrations is complicated by site-specific influences on bioaccumulation. Peterson and Nebeker (1992) observed that the ratio of waterborne Se to tissue Se (i.e., bioaccumulation factors) tended to be similar within a given study site but markedly different among study sites. This may be a result of site-specific factors, such as the waterborne Se form and concentration and food web structure, that affect the degree of bioaccumulation.

The effect of waterborne Se concentration may be significant as Peterson and Nebeker (1992) and Zhang and Moore (1996) noted that there is a negative correlation between waterborne Se and water-to-tissue bioaccumulation factors. As waterborne Se concentrations increase, the proportion of the Se that is taken up by the exposed organisms decreases—the relationship is nonlinear.

In terms of criteria-setting, the implications of among-site variability and non-linear uptake rates are that the use of a single bioaccumulation factor for extrapolation from water to tissue would yield erroneous results.

III. Technical Issues Associated with a Sediment-Based Chronic Criterion

Note: The comments in this section are based extensively upon two publications: Canton and Van Derveer (1997) and Van Derveer and Canton (1997), which discuss the aquatic chemistry, fate, and biological effects of sedimentary Se. Citations are provided for information that is not contained within these publications.

8. Which forms of selenium in sediments are toxicologically important with respect to causing adverse effects on freshwater aquatic organisms under environmentally realistic conditions?

Comments: Dissolved Se has three potential fates in aquatic systems: remaining in solution, absorption by organisms, or association with particulate matter and sedimentation. Sediment is clearly the dominant repository for Se in aquatic ecosystems and sedimentary Se is in the organic fraction (detritus). Detritus can account for >50% of the aquatic ecosystem energy base and, consequently, the sediment to biota pathway is the most important for long-term Se cycling. Particulate Se, as sedimentary, detrital, or suspended Se, has been repeatedly implicated as a causal or contributing factor for food web contamination at sites throughout the U.S.

In reference to development of appropriate water quality criteria for Se, Luoma et al. (1992) reported that “selenium clearly requires a protective criterion based on particulate concentrations or food web transfer”. Likewise, Presser et al. (1994) in summarizing 20 irrigation drainage studies conducted throughout the western U.S., concluded that “the degree of development of organic-rich sediments (i.e., detrital layers), which is known to

accelerate the entrance of Se biologically into the detrital food chain, and anaerobic and reducing conditions for geochemically sequestering it in bottom sediments, must be taken into account for determining the potential for bioaccumulation".

Van Derveer and Canton (1997) reported that the concentration of total sedimentary Se is a reliable predictor of chronic effects upon fish and semi-aquatic birds. Based on a review of 25 study sites located throughout the U.S., they identified 2.5 μg/g as a predicted effects level and 4.0 μg/g as an observed effects level (all sediment concentrations reported herein are expressed on a dry weight basis). Total sedimentary Se concentrations of <1.8 μg/g were consistently associated with an absence of adverse ecological effects (n=11). Total sedimentary Se concentrations of >4.0 μg/g were consistently associated with observed ecological effects (n=7) or predicted effects (n=4). Ecological effects associated with total sedimentary Se concentrations of >1.8 to <4.0 μg/g were variable, ranging from no effect to predicted effects (n=5).

For the purpose of this workshop, which focuses solely on aquatic toxicity, these data were reanalyzed, using the approach described in the original publication, to include only fish. This reanalysis revealed a predicted effect level of 2.4 μg/g and an observed effect level of 4.5 μg/g (Figure 1). These effect levels are consistent with the "low" and "high" hazard levels for reproductive effects in fish and birds proposed by Lemly (1996). The six-month average concentration of sedimentary Se for the central Minnesota experimental streams that were dosed with 10 μg/L of selenite was 2.85 μg/g (Allen 1991). This concentration, which is between the predicted and observed effect levels, corresponded to reduced bluegill survival (Hermanutz et al. 1992) and developmental abnormalities in fathead minnow (Schultz and Hermanutz 1990; Hermanutz 1992).

The clear delineation between no effects and observed effects concentrations suggests that total sedimentary Se is a powerful predictor of adverse effects on fish. Little, if any, data on the direct toxicity of sedimentary Se to aquatic invertebrates are present in the literature. This may not be an impediment to sediment-based criteria-setting, as invertebrates appear to be relatively tolerant of Se exposure, largely serving as a conduit for Se transport through the food web.

Figure 1. Reanalysis of Sedimentary Selenium Toxicity Data from Van Derveer and Canton (1997) Using Only Effects Data for Fish.

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9. Which form (or combination of forms) in sediment are most closely correlated with chronic effects on aquatic life in the field? (In other words, given current or emerging analytical techniques, which forms of selenium in sediments would you measure for correlating exposure with adverse effects in the field?)

Comments: The dominant Se species in sediment are elemental and organic Se, which typically constitute most of the total sedimentary Se burden. Van Derveer and Canton (1997) reported that about 85% (n=8) of the total sedimentary Se content was present in the elemental (mean=42%) and organic species (mean=43%) in streams of the middle Arkansas River basin, CO. Similar sedimentary Se speciation has been reported at Se-contaminated coal-fired power plant reservoirs (i.e., Belews Lake, NC; Hyco Reservoir, NC; and Martin Creek Lake, TX) and irrigation drainage sites (Kesterson National Wildlife Refuge, CA and Benton Lake National Wildlife Refuge, MT. Comparable speciation has also been reported at uncontaminated freshwater lakes/reservoirs (Phillpott Lake, VA and Lake Murval, TX), a salt marsh (Great Marsh, DE), and National Bureau of Standards (NBS) Standard Reference

Material (SRM) river and estuarine sediments (SRM 1645 and 1646).

My Graduate research (Van Derveer 1997) examined the relationship between sedimentary Se concentration and Se accumulation by Chironomidae larvae (midges) inhabiting streams of the middle Arkansas River basin, CO. Larval Chironomidae were used to represent detritivorous benthic invertebrates because of their abundance, wide distribution, importance in aquatic food webs, and demonstrated capacity to accumulate high concentrations of Se. Paired sediment and Chironomidae samples were collected at ten lotic sites along a sedimentary selenium concentration gradient. Surficial sediment samples were characterized through Se and total organic carbon (TOC) analyses of bulk and <63-μm size fractions. Samples of depurated Chironomidae larvae were analyzed for Se concentration as well as taxonomic and functional feeding group composition. Step-wise linear regression determined that bulk sedimentary Se explained the highest proportion of the variance in larval Chironomidae Se accumulation ($R^2 = 0.817$, $p = 0.0003$, standard error of estimate ±1.6 μg/g)(Figure 2).

Figure 2. Relationship Between the Concentrations of Selenium in Bulk Sediment and Chironomidae Larvae in Streams of the Middle Arkansas River Basin, CO.

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The following is the regression model that was developed for Chironomidae Se on bulk sedimentary Se, after correction for transformation bias:

Chironomidae Se = 12.3716 (bulk sedimentary Se)0.7799 * 1.10.

Chironomidae samples were structurally and functionally similar across all sites except one. Collector-gatherers and shredders were the co-dominant functional feeding groups at all sites except one site where predators were dominant. The co-dominance of fine and coarse particulate detritus feeding taxa may have resulted in the superior predictive capacity of bulk sedimentary Se relative to fine sedimentary Se.

A common issue encountered in field-observational studies, such as this, is ascribing causation to correlative relationships. A case for causation can be advanced based on knowledge that (1) Se in sediment is generally associated with detritus (Van Derveer and Canton 1997), (2) most Se in consumer organisms is derived from their diet (Lemly 1993a), and (3) a majority of Chironomidae larvae; including those collected in this study, are benthic detritivores. Thus, the cycling behavior of Se and the feeding ecology of the study organisms support the validity of the empirically derived model of Se accumulation by Chironomidae from bulk sediment.

The quality assurance/quality control information associated with these unpublished data are provided below. Limits of detection for Se in sediment and Chironomidae ranged from 0.1 to 0.2 μg/g and 0.3 to 1.0 μg/g, respectively. Procedural blank Se concentrations (n=11) were below the limit of detection for all but one blank, which was less than 5% of the lowest measured concentration for any field sample or SRM. Selenium recoveries for SRM 1646 (estuarine sediment)(n=7) and SRM 1577a (bovine liver)(n=4) were within the reported or certified ranges. Duplicate sedimentary TOC analyses (n = 2) yielded identical results and recoveries for laboratory control standards (n = 2) were 112.9 and 120.0%.

In priority order, which sediment quality characteristics (e.g., TOC, etc.) are most important in affecting the chronic toxicity and bioaccumulation of selenium to freshwater aquatic life under environmentally realistic exposure conditions? Of these, which have been (or can be) quantitatively related to selenium chronic toxicity or bioaccumulation in aquatic organisms?

Comments: A positive relationship between sedimentary Se and organic carbon or carbon-containing materials (i.e., detritus) has been reported under a broad range of environmental conditions including microcosms; lake enclosures; and numerous studies of freshwater, marine, and estuarine environments. This correlation may be due to many factors including deposition of biogenic particles (detritus) with associated organic Se compounds, uptake of Se by surface biofilms on organic matter, and dissimilatory reduction of waterborne selenate to elemental Se by sedimentary.

Total organic carbon appears to be the most important sediment characteristic affecting chronic toxicity and bioaccumulation of Se because it is highly correlated with the concentration of sedimentary Se, which in turn is a powerful predictor of bioaccumulation and

subsequent effects upon aquatic biota (see comments for question 8). In my Graduate research (Van Derveer 1997, summarized above) sedimentary TOC did not explain a statistically significant proportion ($p = 0.4$ and 0.6 for bulk and fine sediment regressions, respectively) of the Chironomidae Se variance. This may have resulted from the high degree of collinearity between sedimentary Se and TOC—both variables were not required to explain the Chironomidae Se variance. This phenomenon, where sedimentary Se accumulation is determined by the amount of sedimentary organic carbon present, is apparently widespread. With respect to criteria development, sedimentary TOC need not be addressed directly. Rather, the criteria should in some way account for the quantity of sedimentary organic carbon present at a given site.

How certain are we in relating water-column concentrations of selenium to sediment concentrations? What are the primary sources of uncertainty in this extrapolation?

Comments: Van Derveer and Canton (1997) developed an empirical relationship between sedimentary Se and an interaction term of waterborne Se (as mean dissolved) and sedimentary TOC for streams of the western U.S. This model was derived through stepwise regression analysis of dissolved Se, sedimentary TOC, and the interaction term of dissolved Se × sedimentary TOC for streams of Colorado. The resultant model was validated using data for streams in Wyoming, South Dakota, and Nevada. Comparison of the actual and modeled data for the validation sites revealed that the model is a sound empirical representation of the sedimentary Se accumulation process in western U.S. streams. There is a high degree of certainty associated with the ability to predict sedimentary Se accumulation in streams of the western U.S. based on the dissolved Se × sedimentary TOC model.

The sources of uncertainty associated with this relationship include the following:

Specific data for determining the appropriate period of record to be used when averaging dissolved Se data for relating it to sedimentary Se data do not exist. All available data from each study was used in model development.

In some instances, Se and TOC data for bulk or <2.0-mm and <0.063-mm sediment size fractions were expressed as grand mean concentrations due to sample size limitations in the available data sets. Addition of data published since mid-1996 may sufficiently increase the sample size to permit separate models to be developed for each sediment size fraction. Given the information provided above (see question 8 comments) a model that predicts Se accumulation in bulk sediment would likely be of greatest utility for criteria development.

This relationship is not necessarily applicable to streams of the eastern U.S., systems where selenate is not the dominant waterborne Se form, or lentic ecosystems.

Despite the presence of a modest degree of uncertainty, the model described by Van Derveer and Canton (1997) would be suitable for derivation of site-specific chronic water quality standards for western streams or possibly for regional chronic criteria for streams of the western U.S. The uncertainty associated with this approach can be reduced substantially by collecting site-specific dissolved Se, sedimentary TOC, and sedimentary Se data to test model applicability.

With respect to lentic ecosystems, Birkner's (1978) empirical relationship between waterborne Se and organic-carbon-normalized sedimentary Se in lentic systems of Colorado and Wyoming may be suitable for extrapolating between waterborne Se and sedimentary Se.

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Cross-Cutting Technical Issues Associated with Chronic Criterion

How does time variability in ambient concentrations affect the bioaccumulation of selenium in aquatic food webs and, in particular, how rapidly do residues in fish respond to increases and decreases in water concentrations?

Comment: The effect of time variability in ambient waterborne Se concentrations on food web bioaccumulation is difficult to assess and I am unaware of studies that specifically address this issue. Several published studies examine the uptake rates between water and dietary organisms (e.g., algae and invertebrates) and between dietary and consumer organisms (e.g., fish). Data from these types of studies cannot simply be linked together to form a composite response timeline for Se transfer from water to diet to fish. Food web bioaccumulation is most likely a parallel process whereby Se is concurrently accumulating in or depurating from dietary and consumer organisms in response to the Se concentration in the environment. It may be possible to determine a permissible chronic exposure period by reviewing the available literature pertaining to the rate of Se transfer from water to dietary organisms. Consideration should be given to the differential rates at which particular waterborne Se forms (i.e., selenate, selenite, or organic Se compounds) accumulate in dietary organisms (Besser et al. 1989, 1993).

To what extent would the type of ecosystem (e.g., lentic, lotic) affect the chronic toxicity of selenium?

Comments: Inherent differences in Se accumulation and transformation between standing

and flowing waters systems suggest that criteria developed to protect aquatic organisms of one habitat type may be over- or under-protective of the other habitat type. Lemly (1985a) in discussing the 1980 EPA Se criteria criticized the EPA because “...they did not adequately consider differences in the dynamics of Se cycling and the exposure regimes of organisms between lotic and lentic systems...”.

In contrast to standing or slow moving waters, fast-flowing waters have several characteristics that make them less efficient at accumulating Se (Lemly and Smith 1987). These characteristics include the rarity of organic sediment deposits (due to continuous flushing), lower primary productivity, and a greater dependence on terrestrial carbon inputs. As a result, the food web of flowing waters have a reduced accumulation efficiency relative to standing waters.

It can be argued that the connectivity between lotic and lentic systems renders the distinction trivial from a chronic Se toxicity standpoint. Clearly, these types of systems are interconnected; however, there are instances where Se-enriched streams flow into Se-poor streams thus dilution occurs before the Se-enriched water enters a downstream lentic system. In instances where Se-enriched streams do flow directly into lentic systems, it may be feasible to use the Total Maximum Daily Load (TMDL) process to protect a sensitive downstream point.

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